ALZHEIMER'S DISEASE RESEARCH

HEARINGS

BEFORE THE

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INVESTIGATIONS AND OVERSIGHT

OF THE

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SCIENCE AND TECHNOLOGY
HOUSE OF REPRESENTATIVES

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ALZHEIMER'S DISEASE RESEARCH

THURSDAY, AUGUST 30, 1984

House of Representatives,
Committee on Science and Technology,
Subcommittee on Investigations and Oversight,
Johnson City, TN.

The subcommittee met, at 9:52 a.m., in the ballroom of Culp Center, East Tennessee State University; Hon. Albert Gore, Jr. (chairman of the subcommittee) presiding.

Present: Representatives Gore and Pepper.

Mr. Gore. The subcommittee will come to order.

I would like to thank all of our witnesses and all of our guests. I am delighted to see this many people here this morning for this hearing.

I would like to welcome Senator Claude Pepper, who is joining us for this hearing. And I would like to note for the record that Congressman Jim Sensenbrenner, who was scheduled to join us, had a back injury and, at the last minute, could not join us. They had an unfortunate incident, where his house was on fire and he had to jump out the second-story window, and hurt his back. And, at any rate, he could not attend the hearing. But we are delighted that Senator Pepper can. He is the leading authority on aging issues in the United States and the leading authority we have ever had in this country.

I am going to begin with an opening statement, then I am going to recognize Senator Pepper for an opening statement, and then we are going to hear from our witnesses. First of all, an opening statement.

The Subcommittee on Investigations and Oversight today begins 3 days of hearings into Alzheimer's disease. The other 2 days of the hearings will be held in Washington, DC, September 19 and 20.

Public knowledge about Alzheimer's disease is increasing, but, unfortunately, many people do not yet know how insidious the illness can be. It robs approximately 10 percent of the population over 65 of their intellectual ability at a time when they should be enjoying the fruits of a lifetime of activities. As many as 4 million Americans may be Alzheimer's victims, and over 52,000 of them live in Tennessee. Some estimates project the number of victims to double by the year 2000, partly for demographic reasons.

The disease generally progresses in three stages. The first stage is characterized by mild cognitive deterioration and a loss of recent memory. This is followed by further deterioration, including personality changes such as irritability. The final stage may best be described as complete loss of intellectual abilities. The time for this progression may be anywhere from 3 to 15 years.

Presently, the exact cause of Alzheimer's disease is not known, but several possibilities are being investigated by research scientists. And, as of 1 week ago, we have the first promising research lead. The scientist who has discovered this new research lead is going to be testifying on the second day of these hearings.

The research efforts are going to be examined further in the second 2 days of hearings. Today's hearing is going to address the health care issues involved with Alzheimer's disease. In a very real sense, the disease victims include those loved ones who must provide so much care. In the words of a well known book which has just been published about Alzheimer's disease, they provide 36 hours a day of care. Beyond the financial and medical burdens imposed by those who develop this disease are the severe emotional stresses faced by family and friends. The intellectual deterioration of a loved one is a tragic experience that has been described by one family member as a "funeral that lasts for years."

A large percentage of victims live in the community. While there are advantages for the patient to be with the family, there are also disadvantages brought on by the lack of support services. Often, there are few adequate health, social, and personal care services for the patient or the caregivers.

The health care treatment program underway here between the Medical School of East Tennessee State University and the Veterans Hospital at Mountain Home is studying many of these problems. The Office of Technology Assessment estimates that this program here at East Tennessee State is one of only six such programs in the entire country. Whatever knowledge is gained through this pioneering effort will be of little value unless it is passed on to those who need it; and it is, in large part, for that reason that we have invited these national experts to come here to the East Tennessee State Medical School. Witnesses will describe the scope and direction of this program.

In conclusion, we cannot turn our backs on the elderly and their families in their time of need. We must utilize our available resources to find a cure for Alzheimer's disease and to provide adequate health care for the victims. This hearing is designed to provide information that will assist us in ensuring that these goals are achieved as quickly as possible, and that the needs of the victims are met.

And now it is my great pleasure and honor to welcome my distinguished colleague, Claude Pepper from Florida, a friend, the most widely recognized expert on issues involving the aging, chairman of the Rules Committee, former chairman of the Committee on Aging. And I recognize him now for his opening statement.

[Applause.]

Mr. PEPPER. Mr. Chairman and members of the committee, distinguished guests who are to be witnesses today, ladies and gentlemen, maybe the chairman will relent the propriety of his chairmanship and let me sort of explain my tardiness this morning and make my apology for it by saying it sort of reminds me of a story I have heard about back in the old days of the South, when they had dueling.

Two fellows became very much embittered against each other and one of them challenged the other one to a duel. They agreed on the time and place for the duel and pistols as the weapon. The time had almost arrived at the appointed place for the duel to begin. One of the fellows was there with his pistol and his second, but the other fellow had not arrived. Just before time for the duel to begin, a messenger rushed up and handed a note to the duelist who was there from the one who wasn't there. And the note read: "I'm going to be late. Go ahead and shoot." [Laughter.]

Well, I'm delighted to be here this morning. I was very much pleased to meet, as I came into this beautiful auditorium on this very lovely campus, the distinguished president of this institution, Dr. Bella, and to have him tell me that he had his early education and began his life in Florida—Fort Lauderdale, FL. Well I thought to myself, we're just paying Tennessee back a little for Andy Jackson and the Tennessee Volunteers saving Florida from the Indians a long time ago. So, we're delighted to be here in this great institution.

[Applause.]

As a long-time advocate of aid for the elderly, I know what it means to have distinguished and dedicated medical centers such as this where research in problems like Alzheimer's disease, so seriously affecting the elderly are a matter of primary concern. You know, they tell us that one out of—well, the average elderly person in America has about 13 perscriptions a year, about twice as many as younger people or even more. In addition to that, elderly people have to have about twice as much medical care as younger people. So, you can see how important it is to elderly people to have a great institution like this, so nobly and so deeply dedicated to trying to find the cause and cure of so many of these crippling and killing diseases that afflict the elderly people of our country.

When I was born, in 1900 in Alabama, there were only 5 percent of our people who were over the age of 65. Now 11 percent of our people are in that age category. There are 28 million of us. I'm not so old, myself, but I will be 84 on the 8th of September, so I can sort of fairly count myself, maybe, in the category of the elderly. There are now 28 million of us in this great America of ours who are over 65 years of age. And the interesting thing it is, blessed be the Lord for his goodness to us, the group in the elderly group that's growing the fastest percentagewise are the people over 85 years of age.

Sunday, a week ago, I was asked to call a lady in California who was having her 100th birthday that day. They told me to call her between 12 and 1 o'clock. She was a very wonderful lady, had a great record of public service and a great leader among the elderly. I called her at 12:30 p.m. Sunday afternoon.

I got the lady on the phone very promptly. I could tell her voice was a little weak, but she was lucid of mind and very gracious of personality and manner. So, we had a very pleasant talk. I enjoyed the conversation with her. I gave her my warmest congratulations upon being 100 years old. I told her, in the course of the conversation, that I was 83, nearing 84.

Well, just after I hung up the telephone, a telephone call came back to me from that same town in California. It was one of the media telling me that the media was late in getting to the lady's home and they weren't there when I called, would I please call back so the media could take the call. I told them, of course, "I'd be glad to."

And then this fellow calling said, "Would you like to know what this lady said about you when you hung up the phone?"

I said, "Yes. What did she say?"

She said, "You know, that Mr. Pepper sounded like a nice young man." She said, "I just wish I were 83 again." [Laughter.]

But it means so much to the elderly that there are people who care about their problems. And one of the most grievous of all is this problem this distinguished son of Tennessee, this great leader in the Congress, is trying to do something to help the elderly people—all people, for that matter, but his heart seems to be particularly sensitive to the needs of the elderly—we call him Al Gore in the House of Representatives. He chairs this distinguished subcommittee of the Science and Technology Committee. He works in close cooperation with the Ways and Means Committee, which will have to pass on this matter.

Then, eventually, it will get up to the Rules Committee, where the chairman, I can tell you, will look with great sympathy and with expedient haste in giving a favorable response to this legislation when you get your report prepared.

As you know, Alzheimer's disease has now come to be the fourth largest killer of our people in our country. We have found that 1,200,000 Americans have that terrible disease. It is the fourth leading cause of death, as I said, in this country, and accounts for more than half of the admissions to nursing homes. Now, unhappily, our Medicare Program was set up in 1964 and it has not been adjusted to include care for these people who are the victims of Alzheimer's disease. So, there's very little today to which they could look forward by way of assistance in meeting this terrible malady.

We, from time to time, have heard people tell about their problems, who are members of the family of victims of Alzheimer's disease. I remember a lady, not long ago, came into my office who was talking to me. And I said, "What are you doing now?"

"Well," she said, "I was retired but I'm working, now; and it takes every penny I can make to support my husband in a hospital, who has Alzheimer's disease."

And I said, "Well, I'm so sorry to hear that."

She said, "By the way, Mr. Pepper, the other day I had a letter from a lady friend of mine in England. She said her husband had Alzheimer's disease and she said he's in a hospital, but she said it didn't cost her a penny. How is that?"

"Well," I said, "we just haven't developed yet, in our country, an adequate way of dealing with this terrible problem and the terrible problem it presents to the members of the families of these unfortunate victims."

It is a progressive, deteriorative disease of the brain. I saw in the paper the other day, Al, that some scientist had suggested maybe it was the absence of some quality there, maybe some protein or something in the brain. The kind of research that you're going to do here and the kind of research that our bill is going to accomplish of which Al and I are the principal sponsors in the Congress, that's one of the things that we're going to be looking for—what in the world is the cause of this terrible deterioration that all of a sudden hits the individuals in the brain?

Well, in many ways it is one of the worst of all diseases. As Dr. Robert Perry has said, a leading researcher in this field, quote: "All diseases are depersonalizing to some extent, but you are still human, but you'd know one—you can still respond to pain, anger, and hunger"—more or less elemental feeling—"but Alzheimer's victims—takes away," he said "steals man's humanity. It robs him of even the ability to think." The cost of caring for Alzheimer's disease victims is estimated at $17,000 to $50,000 per victim a year.

I heard a lady, not long ago, complaining that she and her sister were supporting their mother who was in a hospital for Alzheimer's disease. And she said, "Within 3 months my sister and I will have spent every dime we have and our family has. We don't know what we're going to do after that."

Most of that cost is borne by family members. Public and private resources available to assist individuals and families of those afflicted with this disease are not available, generally, to Alzheimer's patients and their families. Federal coverage under Medicare and veterans' health programs is minimal. Private assistance through supplemental and other health insurance policies is almost nonexistent. Only Medicaid, of all the public programs, provides significant assistance. Payment of about $6.5 billion a year is paid out through nursing home care of Alzheimer's patients; but the trouble with this is, you have to be utterly impoverished in order to get the benefit of Medicaid. Before you become eligible you have to exhaust your resources and be down to about $1,500 in worth.

Nowhere in our society are individuals left so unassisted and so exposed to the demands of a catastrophic illness as are the victims of Alzheimer's disease. In the words of an Arizona man, the husband of an Alzheimer victim who wrote me in May: "I find it strange that if my wife had a disease from which she could recover or was ill with something like cancer, where she could be placed in a hospice, she could at least be helped a little bit for a few months; but we are told with this disease there is no aid available."

Your subcommittee has been doing a splendid job, Mr. Chairman, in bringing to the people the awareness of the terror of this disease. But we cannot limit our response to a reliance on research and science. We must recognize that Alzheimer's disease reflects the largest single gap in our health and social programs, the essence of a long-term care policy and delivery system, the absence of that policy which would provide help for our victims of Alzheimer's disease. As you know, you and I and some of our colleagues have introduced legislation to begin to address this problem. This bill of ours provides for the establishment of 20 regional centers for the treatment of Alzheimer's disease and similar diseases.

We have, in Miami—my late wife for 15 years was chairman of the Bob Hope Dinner in Miami to raise money for Parkinson's disease. You know that there is a considerable similarity between Parkinson's disease and Alzheimer's disease. Parkinson's disease begins to freeze the muscles. A first cousin of mine, a wonderful lady, was a victim of Parkinson's, and the muscles finally tightened, and tightened, and tightened until finally she couldn't swallow, she couldn't talk, she couldn't move. She finally passed away—the victim of this kindred brain malady.

And, by the way, Mr. Chairman, I'm going to see to it the best I can that very particular consideration in the location of one of these 20 centers in our Nation, under this bill that you and I are the authors of, would be right here at this fine college in east Tennessee, this medical school, that I think would be a good place for it.

[Applause.]

I think it is appropriately located, geographically. I think the kind of spirit here is the spirit that we are looking for among those who are trying to find the cause and cure and provide some program of assistance for this terrible thing.

These Alzheimer treatment centers would be responsible for providing a central facility for the diagnosis, evaluation, and counseling of patients and families of these afflicted with this disease. In other words, the victims in east Tennessee will have some place nearby to come and get care of the sort that would be meaningful to them. They will also develop standards of care, provide training for professionals and nonprofessionals in the diagnosis and treatment of this disease, and develop cost-effective means and methods of treatment of patients who are the victims of Alzheimer's disease.

We are just late, I regret to say, coming to recognize the terrible extent—it's the fourth killer. I guess heart and cancer are the two top, and I don't recall what the third is; but this is the fourth killer of our people. And I hate to say so, but it's a terrible death that it inflicts upon its victims.

It is our intent, as you know, that these centers provide comprehensive treatment for Alzheimer's patients, embracing both acute and long-term care. I am chairman now of the Aging Committee of the Subcommittee on Health and Long-Term Care. Long-term care is one of the great needs. We would be providing long-term care through this center, here, if it were established here, for the victims of Alzheimer's disease who don't have such benefits available now. It is our intent that these centers provide comprehensive treatment for Alzheimer's patients, embracing both acute and long-term care, I'd say, and supported by a per capita payment to the center based on the combination of benefits to Americans, benefits to which they would otherwise be entitled under Medicare, Medicaid, and other medical acts, and the supplement of a million dollars a year appropriation for each one of these centers, to support teaching and training of staff and research. I believe it is time that we come to grips with this terrible enemy of our people, Alzheimer's disease.

I want to commend you, Mr. Chairman, for taking the initiative in this fight, showing how concerned you are about this problem. Your colleagues in the Congress are going to respond to your sentiment and to your leadership. And you know I can be surely counted upon, every step of the way, to do with you what we can for these great and good people who deserve help from this terrible enemy which afflicts them. Thank you very much.

Mr. GORE. Thank you, Mr. Chairman.

[Applause.]

Mr. GORE. Thank you very much, Mr. Chairman, for your statement and for your kind words, and also for the exciting words about the possibility of locating one of these 20 centers at the Quillen-Dishner Medical School in East Tennessee.

I'd like to ask unanimous consent at this point for the record to be held open to include the opening statements of Mr. Sensenbrenner and, should they desire to have one, Mr. Skeen and Mr. Winn. The ranking Republican member of the full Science and Technology Committee, Mr. Larry Winn, had a family member, his father, who died of Alzheimer's disease, and in many ways this 3-day hearing is a joint effort with Mr. Winn, who is extremely interested and concerned that the Science and Technology Committee do a thorough, thorough job of getting to the bottom of this matter and making certain that we're moving as a country in the right direction.

Now let me ask our first panel of witnesses to come to the witness table.

Dr. Edith Summerlin, who is dean of the School of Nursing here at East Tennessee State, if you would come forward to the witness table and have a seat right there, and Mrs. Beatrice Abernathy of Kingsport, TN, and Mr. Tommy Dillow of Kingsport, TN, if both of you could join us at the witness table. And while you're coming to the witness table, let me say something I neglected to say earlier.

I want to express my thanks and the thanks of the subcommittee to the staff, faculty, and administrators of East Tennessee State University and of the medical school for their assistance and cooperation in arranging this hearing. We're most grateful and I want to tell you how grateful that the subcommittee is.

Now, before going to these witnesses, I thought it might be interesting, Mr. Chairman, if I could ask those members of the audience how many of you—if you don't mind me asking—if you could answer with a show of hands, how many of you have a family member or a close friend with Alzheimer's disease or who has, perhaps, died from Alzheimer's disease in the past. Could I see a show of hands?

[Show of hands.]

Mr. PEPPER. My goodness alive.

Mr. GORE. Thank you very much. I think that's an indication of how important this issue is. 26,000 of the Tennesseeans who suffer from Alzheimer's disease are located here in east Tennessee.

Well, enough of this. Let's go on to the witnesses. And all three members of our first panel have had a personal experience, as you will see from their statements. But without any further ado, let me go to our first witness, Dr. Edith Summerlin, who is dean of the School of Nursing here at East Tennessee State.

Dr. Summerlin, I am delighted that you could lead off our hearing. And please proceed with your statement.

STATEMENT OF EDITH BUTON SUMMERLIN, PH.D., DEAN, SCHOOL OF NURSING, EAST TENNESSEE STATE UNIVERSITY; ACCOMPANIED BY MRS. BEATRICE W. ABERNATHY, KINGSPORT, TN; AND TOMMY ALAN DILLOW, KINGSPORT, TN

Dr. **Summerlin**. Thank you very much. I want to thank the commission for giving me this opportunity to share with you my personal and professional perspective about some of the needs and problems of a caregiver of an Alzheimer's disease victim.

Although my father has been disabled for 32 years, my mother, the primary caregiver, has provided what care he needed without problem or complaint. It has only been over the past 5 years that she has begun to falter in her ability to cope, and it wasn't until March of this year that a label, Alzheimer's disease, was given as a medical diagnosis for the drastic changes in my father's behavior and physical condition.

Prior to his diagnosis, and because my parents have resided with me—I'm sorry.

Mr. **Gore**. No; you take your time. That's fine, Dr. Summerlin. I should have noted in your introduction that not only are you dean of the school of nursing but one of your parents has this disease; and your perspective is of great interest to us in understanding this matter. And I'll talk on a little bit more if you want me to.

Dr. **Summerlin**. No; that's fine. I think I'm ready.

Mr. **Gore**. You might bend that microphone down a little bit, that way.

Dr. **Summerlin**. Thank you. I've been called on by my mother to provide support and professional knowledge as a registered nurse in the following ways: one, to control my father's verbal and physical abusiveness; and two, to assist in providing physical care, such as demonstrating to my mother how to feed my father, how to get him to take his medication, how to bathe him and massage the reddened areas so his skin would not break down, how to lift him in and out of bed, and how to get him up in bed—to just mention a few.

In addition, I have tried to explain his disease to my mother; which she still doesn't accept. I have tried to help her develop her independence and coping skills for each day as well as in preparation for what the future is to bring.

My mother has never driven a car, so I or one of my children provide all transportation for her to accomplish any shopping or getting about that she must do. I have tried to get her to recognize her need for rest and relaxation.

I keep my brother, who lives in New York, informed periodically about our parents as well as other distant relatives who call to speak to me because I am the nurse and, supposedly, the knowledgeable person.

I believe I have experienced each of the five stages of reaction that Dr. Tensik and Ms. Maylor speak about, and these reactions are: initial denial, over-involvement with the patient, anger, guilt, and, hopefully, resolution or acceptance of the disease. But I still experience frustration and helplessness. When I visit my father and he doesn't recognize me, when my mother still asks me why does he treat her the way he does, when I don't seem to have the knowledge, time, effort, or energy to cope with all the stress and strain of being the professional at all times—I'm sorry.

Mr. **Gore**. No, that's—if you want to take a short break, that is fine.

Dr. **Summerlin**. No; I wonder how well the family would be doing if I weren't a professional nurse.

Needless to say the problems and needs are many and I have only highlighted a few. Basic to all is the need for research and more knowledge about the cost, treatment, and prevention of Alzheimer's disease. The psychological, physiological, cultural, social, and economic ramifications of the disease on families needs to be studied. The impact that the increasing numbers of elderly, the changing roles of women, and the decreasing numbers of children in families will have on society and its ability to care in the future for Alzheimer's disease victims, needs to be investigated now and some steps taken to plan for tomorrow.

Thank you again for this opportunity, and if I can be of any further service let me know.

[Applause.]

[The prepared statement of Dr. Summerlin follows:]

TESTIMONY FOR CONGRESSIONAL HEARING
ON ALZHEIMER'S DISEASE

August 30, 1984

ETSU CULP CENTER BALLROOM

I am the only living daughter of an Alzheimer's Disease victim. My Father, who is 81 years old, now resides in a nursing home. For the past five years, my Father and Mother have resided with me and my children. In February 1984, my Father was hospitalized for 21 days for a urinary infection. At this time, based on the history of my Father's behavior and his deteriorating physical condition, our family physician ordered a battery of tests to be done which rule out all possible causes for my Father's behavior and condition. In March, the diagnosis of Alzheimer's Disease was made. Home health care was discussed by myself and the social worker from the hospital, however, the family physician did not see this as a viable alternative, thus, a decision had to be made regarding a nursing home placement or a return to my home. The decision was made by my Mother that she could no longer care for him in the home with or without my help and, therefore, nursing home placement was sought. We were told my Father was not eligible for placement at the Veteran's Administration facilities.

All of this may sound simple and even perhaps heartless to anyone who isn't familiar with the situation but many hours and painful discussions preceded this step.

My Father, a native West Virginian, dropped out of school in the eighth grade. At the age of seventeen he enlisted in the Army and served as a lead team driver, during World War I, for the 16th Field Artillery Unit at Ft. Sill, Oklahoma. He was kicked by one of the horses and later, after his discharge, he had to have a kidney removed in 1927 due to an infection. After working at odd jobs, he settled on being a bee keeper's assistant until after the depression and then turned to being a garage mechanic. He remained at this until September 8, 1952, when, at the age of forty-nine, he had an automobile accident in which he broke his neck and became disabled and unable to be gainfully employed from that day on. For years my father has been under regular medical supervision. The first mention of Alzheimer's was in March, 1984. His sole income since the accident has been social security disability.

My Mother, then forty-four years old, became the care giver and only gainfully employed member of the family. She had dropped out of school in the eleventh grade to take care of her siblings and had never been employed outside the home. She went to work in a cafeteria in a plant in our hometown in Ohio where she remained employed until she reached retirement age and, thus, has her own social security benefits.

My brother, studying to be an electrical engineer at the time of the accident, had his own family which has expanded to six children since then--one of his children is mentally retarded with Down's Syndrome. He lives in New York. He has been to visit once in the past five years and rarely calls or writes to our parents. He is aware of our Father's diagnosis and condition and has chosen not to visit or contribute support of any kind.

I am a professional registered nurse. I have served my country as a U. S. Air Force Nurse and am currently a U.S. Army Reserves Nurse. I have worked as a community/public health nurse in the Washington, D.C. area and for the past seventeen years have been involved in nursing education. I have four children ranging in age from 15 to 22. I have been divorced for nine years and have received no support from the children's father. When I accepted the position of Dean of the School of Nursing at East Tennessee State University and was making the move from Ohio to Tennessee, I made the decision to move my parents with me into my home. My Father was then seventy-six and able to get about slowly but without assistance, he was in control of his bowels and bladder, able to feed himself, able to assist in dressing himself and able to communicate with the family. In 1980, shortly after our move, he had to have surgery for cancer in the rectal area. It was at this point that drastic changes in his behavior began to occur or, at least, that is when I became aware of his behavior. Had I realized what was to come I don't believe I would have moved my parents to Tennessee with me. I would have encouraged them to move from the home they had built with their own hands, and in which my brother and I were born, to a senior citizen's center within the city limits and somehow we would have found the financial means to accomplish this. I believe my Father would have been "better off" among friends and family. But more importantly, my Mother would have been near friends and family to give her the emotional support she needs and relief from the tedious care giving tasks which increased over the past five years. My Father progressed from the use of a walker to a wheelchair to a bedridden status. He went from feeding himself to being fed. He went from assisting with dressing to having to be dressed. He went from being able to get to the bathroom by himself to using facilities at the chair or bedside to incontinence. He went from being a social, laughing, good natured individual to one who was abusive and hostile verbally and physically. He went from sleeping normally to sleeping all day and harassing my Mother and family all night. He became suspicious of everyone and everything that went on around him. It was no longer possible to carry on a conversation of any kind with him and he no longer recognizes me or the children. For several years, he has been on medication to control his abusiveness.

Many of the characteristics of an Alzheimer victim have been masked by my Father's disability.

As these changes occurred with my Father, my Mother began scape goating my two boys. I became a referee in the almost daily shouting matches which always seemed to occur when I would arrive home from work. One of my daughters, on the other hand, was used by my Mother as a sounding board from all that was wrong with the rest of us and was made to feel guilty and, thus, manipulated into taking my Mother's side. The end result was a destruction of family unity and calm which I had worked hard to build with the children and myself over the previous four years. In addition, I saw my Mother age rapidly. She would cry easily, something I had never observed before. She gained weight and allowed her physical appearance to decline. She had enjoyed going out to the store at least three times a week as the only outlet or escape but as my Father's condition worsened, she gave up competely this activity. She has continued to deny my Father's condition and believes he is being "nasty" on purpose. Denial is a way of life for my Mother. This seems to be her way of coping. My Mother is now living in an apartment next door to the nursing home where my Father is residing. This move was essential so she could visit daily. Before she moved, I was coming home every day at noon to pick her up and take her to the nursing home and then picking her up on my way home from work. This became extremely difficult for me with the many meetings and scheduling conflicts it created revolving around my work or the children's activities. It was also very hard on her because she had to wait for me, whereas, now she can come and go as she needs to or wants to depending on my Father's behavior or condition.

My oldest child, a girl, was no longer in the home when we moved so she did not experience personally any significant problems. My second child, the daughter used by my Mother, chose to go to college in Ohio to get away from the everyday conflict in the home and did not visit as often as she might have had circumstances been different. Now graduated from college, she has taken a position out of town. My third child and oldest son, now nineteen, graduated early from high school and immediately went into the Army instead of going to college as planned. He is home now, employed and in his own apartment. My fifteen year old son has probably suffered the most because he was in the situation the longest. He has lost essentially all respect and love for both his grandparents because of their behavior. My son would leave the house for school and remain away until after I would return from work. He especially would avoid having to stay home with his grandfather when my Mother and I would go to the store. He had to carry my Father to bed several times before my Father was hospitalized and this distressed him. As a result, I have had to coerce each of the children to visit either of their grandparents. Neither the children nor I would have friends over because of my Father who would sit in the front room with his pants unzipped and/or his shirt unbuttoned or he would cry as he spoke about the past in response to any comments directed to him. People on T.V. would be identified as relatives or friends by him, thus, embarrassing all of us. It was simpler to not encourage visitors.

Alzheimer's Disease, although known about since 1907, has received little serious attention until recently. When my Father was diagnosed in March of this year as having Alzheimer's Disease, I naturally turned to nursing literature and textbooks being used for instruction in the education of registered nurses. What I found was shocking. The textbooks I consulted either didn't mention it by name or mentioned it only briefly and the few words that were there were often in conflict, e.g. on one page "occurs primarily" in women vs. "it affects men and women equally" in another section of the same textbook written in 1983. In a textbook of 700 pages, written in 1981, Alzheimer's Disease was mentioned in one paragraph and research from the National Institute of Health (1979) was sited with the promise of more research to come to provide an understanding of the etiology, treatment, and prevention of Alzheimer's. Another textbook of 400 pages written in 1982, had a total of four paragraphs on one page. A 2000 page textbook written in 1983 had citations on three pages regarding Alzheimers. When this proved futile, I then consulted physicians in the community and they weren't able to give me the answers either that I sought regarding the disease.

Based on the previously noted experiences, I make the following observations:

1. It is clear to me that accurate, up-to-date knowledge is needed for both professionals and lay individuals regarding signs and symptoms of Alzheimer's Disease and its impact on the family and the health care system.
2. Much of what is being done to help care givers is after the diagnosis is made and for me and my family that was far too late. All of the heartache, decision-making, and realization of what was and would be had been resolved.
3. There are still too many unanswered questions regarding the cause, prevention, and treatment of Alzheimer's which only research can answer. If one looks at the various theories being advanced as to cause/effect my Father's history fits some of them and may or may not fit others. Who is to say? Will I or my children become victims? If so, what, if anything, can be done to delay or prevent its happening?
4. The education of medical and allied health personnel is sadly lacking if the textbooks used in nursing are any example of how little is taught and how conflicting the messages are in what is presented.
5. Financial resources are sadly lacking to assist families in meeting the cost of nursing home care since medicare/medicaid do not cover this diagnosis.
6. What kind of tax relief can be offered to the already over-taxed middle man who seems to only get hit harder and harder?

I feel I have gotten out from under the daily pressures associated with my Father's care. I am not called on to help, except occasionally, with his physical care. The financial burden of providing housing, food, electricity, and incidentals to accommodate my Father's disability and Alzheimer's Disease are no longer present and, therefore, I have my home up for sale with the hope that

I will replace it with one which meets my needs and is not so costly. I am rebuilding a social life and reclaiming my home as my own domain. I no longer live in one room of the house buried in work or books. The emotional drain of trying to juggle everything and keep peace in the family is no longer a problem, so for me life is starting all over again. I will enjoy it while it lasts because I can see the day ahead when my mother will no longer have any money, her health will fail and it will be her turn to be cared for and it will start all over again.

Thank you for allowing me to share with you some of my personal experiences and reactions to living with an Alzheimer's Disease victim.

Edith B. Summerlin, R.N., Ph.D.
Dean, School of Nursing
East Tennessee State University

My name is Edith Buton Summerlin. I was born February 23, 1935 to Ruth Wilson Buton and Earl Cutting Buton in Tiffin, Ohio. I grew up in Tiffin, Ohio. I lived three miles out of town but attended city schools. I spent my spare time playing french horn in the band, reading books, and studying. Upon graduation from high school, I attended The Ohio State University. I worked two-three jobs while in college. I graduated from OSU with a baccalaureate degree in nursing in 1957.

While waiting to hear whether I had passed the licensure exam to practice nursing, I worked as a private duty nurse and as night nurse for one month in a children's convalescent home. Once I was notified I had passed and was a registered nurse, I entered the U.S. Air Force as a second lieutenant. I was stationed at Keesler Air Force Base in Biloxi, Mississippi, until my discharge three years later at the time of my marriage. My husband and I moved to the Washington, D.C. area where we lived for fourteen years. While there, I worked as a public health nurse in Washington, D.C. and Prince George's County and taught public health nursing at The American University and Towson State College. During this time, I completed my master's degree at The Catholic University in nursing (1967) and I had four children. Ruth was born October 25, 1961, Robin was born October 16, 1962, Dennis was born October 8, 1964, and Robert was born January 22, 1969.

The children and I moved from the Washington, D.C. area in 1974. I was divorced in January, 1975. My ex-husband was a child and wife abuser. The 1974-1975 year was a very difficult year for me and the children. Ruth broke her arm, Robin broke her leg, Dennis had dental problems and Robert got pneumonia. Somehow we managed to survive.

In 1975, we moved to Ohio. The children were very active in school activities and I was consumed with my position at The Ohio State University. I completed requirements for my Ph.D. from The American University in 1976.

I joined the U.S. Army Reserve Nurse Corps in 1979 for supplemental income and retirement benefits. I am now a Lt. Col. in the 912th MASH Unit.

Professionally, I am active in the professional nursing organizations, the March of Dimes and the Johnson City Hospice.

My position as Dean of the School of Nursing at East Tennessee State University fills many of my remaining hours and has since 1979.

Someone recently asked me what I do for me and my answer is I still like reading and music. I have also developed a liking for backpacking which I try to do at least once a year. And, of course, I participate in my children's activities.

Mr. Gore. Thank you for your statement and for your courage. We appreciate all that you do.

The full text of all our witnesses' statements will be included in full in the record.

We're going to hold up on questions until all three of these members have testified.

Mrs. Beatrice Abernathy from Kingsport, TN, we thank you for your willingness to appear here today and we'd like to hear your statement now.

Mrs. Abernathy. I share a similarity to my friend here, in that I had been a nurse. My background was with the elderly.

Incidently, Congressman Pepper, my long-time hero, Charles Lindbergh, I've let him go. You're my hero——

[Laughter and applause.]

Mr. Pepper. Thank you very much.

Mrs. Abernathy [continuing]. Right along with that glamorous Rita Hayworth.

I wanted to tell you today that Alzheimer's life style is not shown on Dallas, it's not shown on Falcon Crest, not even on Leave It To Beaver. The multimillionaire Ewing family, those corporate executives—bed hopping, but they never get V.D. They drink continuously. They never use R.A.A. clinics. If they go bankrupt they change the script. We don't need the V.D. clinic. They are used and there are many. At our house we don't need an A.A., but I can't find a clinic and I can't change the script.

Back at our ranch, or our Alzheimer's house, we live an entirely different life. I run around barefooted because I can't have the Morton's neuroma removed from my foot. It would be 6 weeks. I lose my balance too much already. I dress in baggy, comfortable, work-hard clothes, so that I can tug, and pull, and stretch, and bend. My husband, Ab, doesn't wear that beautiful blue oxford cloth shirt any more, nor a tie, nor his favorite tie clasp. He can't carry his wallet or his favorite knife in his pocket.

We no longer have an attractive bedroom. Ab sleeps in a hospital bed. He is secured in it by a strong safety belt. The side rails are up. Actually, we don't have a dining room any more either; that's Ab's room, and it's complete with a suction machine, a potty chair, and hundreds of sheets, diapers, plastic pants, peroxide for mouth care. That room is convenient to the kitchen. That's the room that is convenient to that old washing machine.

I really want my husband back. He is almost mute now. There will be weeks when he doesn't say anything. I'd like to talk to him. I'd like a hug from him. If I tell Ab: "I need a hug," he might say:, "I have another pair of black shoes." He might be mute for 1 week or 2 weeks, but then he shocks me by a very lucid comment.

Just about when I'm ready to accept, he's got to go to a nursing home. The quality of his life is now nonexistent. He probably doesn't know me any more. That very morning he'll say: "Hi, Honey, did you sleep OK?" It's erratic. He might even try to explain: "Somehow, I can't talk." He does smile at me occasionally. He seems comforted when I am there. He sometimes hits me because he doesn't know me. But I would like him back.

I really don't know what to do financially. I ran the heat pump filter completely backwards for months. I don't know what to do about our insurances, and I'm certainly in a total mess legally. We've passed through the frightening stage of flooding the bathrooms when he carried around his gun, his sudden combativeness, his imagined fires, those explosions he saw that terrorized him. I think this period was perhaps, maybe, the most devastating. I don't know what else might happen.

I believe he knew he had lost control. I believe he knew he couldn't protect himself or me. I believe he was overwhelmed. He must have suffered dreadful agonies at this time, because he would tell me: "Now, just be real quiet, I have a thought." Later he would say: "I lost it."

One time he asked me to come back to the bedroom and close the door, that we needed to talk privately. I closed the door, and his soul cried: "Am I losing my mind? Honey, am I going crazy?"

I'm glad he asked me. I assured him that this was an organic brain disease. I tried to assure him—oh, Lord, I've lost my place here.

This disease is so erratic; it is the funeral that never ends. I want Ab back. I hate the isolation. Rarely does a friend call, rarely does a friend come by. If I'm at the grocery store I'm either avoided or somebody one ups me—"Hi. Bless your heart. Did you know about Mrs. so-and-so with that terrible, terrible crushed hip?"—as if it's necessary to tell me something worse, might console me. I lived my former husband's year of cancer. I lived my aunt's months of cancer of the spinal cord. But Alzheimer's disease goes on and on—4 years, 10 years. Duke University tells me it can go on for 20.

A renowned psychiatrist recently, specializing in Alzheimer's disease, told me I caused the isolation, myself, that I should put him in a nursing home, go out, look for another husband; people didn't want to be around me because I was too good, I was too devoted. I don't agree. I don't like being in this place. We human animals were endowed with the highest of intelligence but we avoid those that are different from us. We want to look the other way. The cow can desert her defective calf, the dog and cat destroy their defective offspring; but surely we're more highly educated. We can't be that animalistic, but we do avoid and we do look the other way.

I must say there have been some improvements in attitudes toward the dying cancer patient; we do have hospices. We have Ronald MacDonald Houses for Children. But even the well elderly are avoided. I hate to announce this public, but did you know that on the new Tennessee driver's license those wonderful photographs for identification, you don't have to have them if you're 60. That's what it says. Is it also saying we're nonpersons over 60?

Attitudes have just got to change. We may fantasize with the "J.R.'s" and the "Sue Ellen's," but don't tell me I have to hide my husband. My grandchildren would like more of my time and my attention. I'd like to be able to give it to them. But at least Ab has gifted them with this real world. We do get old and we may become ill. My 2-year-old grandson, Julian, is not afraid of Ab. He'll run his car up the veins of his arms. Once in a while Ab will smile at him. Julian is not afraid. But other children scream, cry, run when they see my husband's blank stare or his choking noises.

Why can't I keep my husband at home? All my friends say: "Put him in a nursing home." His sister says: "Put him in a nursing home." His beloved company would be happy to have him in a nursing home. I've noticed the great big sign Abernathy-Thomas Engineering Co. is gone. It's been painted over for 2 years. Are these beloved employees waiting for him to die?

Why does everyone want Ab in a nursing home? No one has said: "We'll start a nursing home fund." I think they want to avoid. I think they would like to look the other way.

I am angry. Most of the time I am very frightened. I am isolated and I am alone.

How can I keep my husband at home? Not by Medicare, not right now by Medicaid, not by medical insurance. What choices do I have? Remember, I can't ask my husband.

Our Blue Cross and Blue Shield policy states: "You may have a nurse 8 hours a day if ordered by a physician." Indeed, it was ordered by the physician. It was OK'd by the local Blue Cross, it was OK'd by the Blue Cross of Tennessee and Chattanooga. The nursing service double checked that they'd be paid by Blue Cross if we used the nurse.

We got the nurses, Blue Cross paid, and—you've guessed it—we started a hellish merry-go-round. We were qualified, we were disqualified, over and over again. They even agreed that they were wrong; they now would pay 100 percent for the rest of the year. I had overpaid.

You're right: They demanded their first payment back, a few thousand more dollars, and we are in a lawsuit because they say I must pay for the nursing service $9,000 or $10,000. And, incidently, we are in a lawsuit with my husband's own company, that he formed, because we couldn't find out—we were ignored by letters asking for the value of his stock so it could be sold, so we could take care of ourselves.

I mentioned we couldn't use Medicaid, but it won't be long. But, then, Medicaid will have two clients, my husband and myself, unless I go to work, too, at 65 or 70. Public health can give me a nurse on short visits to demonstrate how to do things for my husband. I've checked nursing service that Medicare will pay 3 hours for 3 weeks; that's it. They, too, will show me how to take care of my husband.

I really do realize that I can't have my "Mr. Nice Guy" back. I know that. But who can help me? He needs 24-hour care. At this time I still give 16, sometimes 14, myself; but I need help at least 8 hours. I'm only one of the other victims. If there is 2.5 million Alzheimer's victims and we, also, are victims, we're talking about 5-6-million victims of Alzheimer's disease.

We plead for your assistance. Some suggestions that I had thought of, that will be repetitious, is that region 1 here in upper east Tennessee has eight counties—first, educate through your medical societies updated symptoms of Alzheimer's disease. Then do a survey and locate them. They're not all in nursing homes, they're hidden at home.

Grant funds to public health or to the VA Center to educate home health aids.

Grant us custodial care under Medicare. I have five grown children and eight grandchildren, so please don't take it away from Social Security.

Establish a nonprofit Rita Hayworth House, Claude Pepper House, an Albert Gore House, an Ab's House, but give us a house for these people. Perhaps it could be patterned somewhat like a Ronald MacDonald's. It could be staffed by medical students, student nurses, vocational nurses. And let us help. We do know how to do it. Maybe it could be supervised by the new gerontology department here. The above-mentioned home could be a clearing house for Alzheimer's information. It could be a training facility for physicians, nurses, social workers, and for us.

It has been a privilege to be allowed to express my thoughts. I'm encouraged by the amount of money given to research. I'm grateful for your concern. We can only find honor in our loss by being of help to you. Thank you.

[Applause.]

[The prepared statement of Mrs. Abernathy follows:]

Testimony from Bea Abernathy

An Alzheimer's life style is not shown on "Dallas," "Falcon Crest" or "Leave It To Beaver."

The Ewings are multi-millionaire corporate executives -- bed hopping (they never have V.D.) -- drink continually (they never go to A.A.) -- go bankrupt but a quick change in the script saves them.

However, back at the Alzheimer's house, I live a very different life. I go barefooted. (I can't take six weeks off to remove that Morton's neuroma.) I dress in baggy, comfortable, do-hard-work clothes, to bend, stretch, pull and tug. Ab doesn't dress like J.R. any longer. No blue oxford cloth shirt, tie, his favorite tie clasp, watch, wallet and so forth. He is in washable running shorts, wash and wear shirt. We no longer have an attractive bedroom. Ab sleeps in a hospital bed, secured by a strong safety belt and the side rails are up. Actually, we no longer even have a dining room -- it's now Ab's hospital room, complete with suction machine, potty-chair, and mountains of sheets, diapers, plastic pants, peroxide for mouth care, and so forth. It's convenient to the kitchen and it's convenient to the washing machine.

I want Ab back! He is almost mute now. I want to talk to him. I want a hug from him. One time I told him that I needed a hug and he told me he had a pair of black shoes. Though he may be mute for a week, he then shocks me with a lucid comment.

When I'm just about to accept that he really doesn't know me;

the quality of his life is nonexistent and perhaps it's time for a nursing home, he may well ask, "Did you sleep well last night, honey?" Or he might even explain that he can't think how to talk. He does smile at me occasionally. I know he is conforted by my presence. (He sometimes hits me if he doesn't know who I am.)

... but I want Ab back. I need him: what to do, financially; about the heat pump; and how about insurances? what to do legally?

We've past through that frightening stage of flooding bathrooms, carrying his gun, sudden combativeness, imagined fires and explosions that terrorized him. I think this period was the most devastating (what else could happen to us?). I believe he knew he had lost control, could no longer even protect himself or me, and was completely overwhelmed. He must have suffered dreadful agonies. He would tell me to be real quiet because he had a thought -- but then, "I've lost it." One time he asked me to come in the bedroom and close the door because he needed to talk privately. His soul cried and finally he asked, "Am I losing my mind?" Do most victime agonize that fear and dare not ask? I'm glad Ab did. After much assurance, he wasn't mentally ill but was experiencing an organic change and with an infant dose of Holdol we finally passed through that nightmare. This disease is so erratic, so long lasting, it is truly "the funeral that never ends."

I want Ab back -- I hate the isolation! Rarely does a friend visit and rarely does one call. If I'm at the grocery store, I'm either avoided or quickly told of Mrs. So-and-So's crushed hip, as if it's a one-upsmanship game.

I lived my former hsuband's year of cancer ... I lived my aunt's months of cancer of the spinal cord. Alzheimer's disease can continue four years, ten years and even 20 years -- and at no stage can Ab prepare to die.

A renowned psychiatrist, specializing in Alzheimer's, told me I caused the isolation; put him in a nursing home, look for another husband. People didn't want to be around me; I was too good, too devoted. But I don't agree. I don't even want to be in my place.

We human animals were endowed with the highest intelligence, but we all avoid those different from us. The cow deserts her defective calf; the dog or cat destroy their defective off-spring. We can't be that animalistic but we avoid and we do look the other way. I must say, there has been some improvement in attitudes toward the dying cancer patient; we do have hospices. We have Ronald MacDonald houses for children. Even the well elderly are frequently avoided. The Tennessee Driver's License now has photos for I.D.'s; but if you are 60 or over you do not need a photo. Why? Are you a nonperson at 60?

Attitudes must change! We may fantasize with the J.R.s and Sue Ellens, but don't tell me I have to hide Ab. My grandchildren would like more of my time and attention, but at least Ab has gifted them with the real world. Two year old Julian is not afraid of him. Ab sometimes smiles at him. No, Julian is not afraid; but I've seen other children freeze or cry at Ab's blank stare or his choking. Why can't I continue to keep Ab at home? Our friends say, "Put him in a nursing home." His sister says, "Put him in a nursing home." His very own company would certainly prefer him in a nursing home. The sign, "Abernathy-Thomas Engineering Company" has been covered by paint for over two years. Are those beloved employers waiting for Ab to die? Why does everyone want Ab in a nursing home? Not one has said, "We'll start a nursing home fund." No, they want to avoid, they would like to look the other way.

I am angry -- I am frightened and I am isolated. I am alone.

How can I keep Ab at home? Not by Medicare, nor Medicaid; not by medical insurance. What choices do I have? Remember, I can't ask Ab. Our Blue Cross/Blue Shield policy states, "You may have a private duty nurse eight hours a day if recommended by a physician." Indeed, our physician recommended it. I was assured by the local Blue Cross office, the home office in Chattanooga, and also that the nursing service was assured. Likewise, those conditions were met. Nurses came and Blue Cross paid; however -- you guessed it -- we were now on a hellish merry-go-round. We qualified, we were disqualified, over and over. At one point I was even told Blue Cross would pay the 100% (I had overpaid my share) for the remainder of a year's time. But alas, finally Blue Cross demanded the payments they had made be returned and I would have to pay approximately $9,000 to the nursing service. A few more thousand dollars and we are involved in a law suit. (Incidentally, there is another law suit with his own company just to find out the true value of his stock -- so that it can be sold.)

I mentioned we couldn't use Medicaid -- true -- not at present, but it perhaps is not too far off. When we are down to Medicaid, they will have two clients. Or do I go to work at 65 or 70?

Public Health can send a nurse for brief visits or show me how to use equipment or how to give him an enema and so forth. Home Nursing Service tells me they can instruct me three times per week for three weeks only.

I really do realize I can't have my Mr. Nice Guy back, but who can help me? Ab needs 24 hour total care. At this moment I can give 16 hours; but who can help for eight hours?

I am only one of the "other victims." If there are 2.5 to 3 million Alzheimer's patients, we (the other victim) bring it to 5 - 6 million. We all plead for your assistance.

Some suggestions I've thought of:

1. Region I (eight counties). First, educate through each Medical Society the updated symptoms of Alzheimer's. Follow by a survey to identify the number of patients thus incapacitated.
2. Grant funds to public health to educate home health aides in the care of the Alzheimer's patients.
3. Grant us custodial care under Medicare. I stress, not from Social Security.
4. Establish a nonprofit "Rita Hayworth" or "Alzheimer House" or an "Albert Gore House" in each appropriate region. Perhaps these could be patterned somewhat like a Ronald MacDonald home: staff and student nurses, medical students, spouses

sharing. Supervised by whom? The Gerontology Department at ETSU or Murfreesboro? Again, Medicare would establish custodial benefits.

The above mentioned homes could be clearing houses for Alzheimer information and training facilities for physicians, nurses, social workers and for families.

It has been a privilege to be allowed to express my thoughts. I'm encouraged by the amount of money given for research. I'm very grateful for your concern and interest. We can only find honor in our loss by being a help to you.

Thank you.

BIOGRAPHY OF BEA ABERNATHY

I am Beatrice W. Abernathy, born in North Stonington, Connecticut. Lived in this small village with my aunt (an elocutionist) and uncle (piano builder, politician). Exposed to education, the arts, nature, travel and politics. Very healthy childhood. My wise grandfather lived with us in his later years.

Education: Public schools in North Stonington; Dudley Wheeler Prep School in North Stonington; received R.N. from Sanders School of Nursing, Knoxville, Tennessee, in 1946.

Work: Staff nurse, Oakridge Hospital, Oakridge, Tennessee; School Nurse, Kingsport City Schools, Kingsport, Tennessee; Holston Valley Community Hospital, Kingsport, Tennessee where I worked in the Emergency Room, Psychiatry, was Evening Sueprvisor; Supervisor of Wilcox Hall (Intermediate and Skilled Care); and was Evening Supervisor at Wesley Woods (an intermediate care facility) in Atlanta, Georgia, while husband acquired Ph.D. Chemistry at Emory University.

Marriage to Kent C. Brannock, 1947; 5 daughters. Family life was boisterous and diverse. Outside activities were Girl Scouts; Women of Kingsport Symphony; St. Paul's Episcopal Church; teaching in Holston Valley Community Hospital Clinic; three years at Atlanta. Unfortunately, the marriage dissolved in 1968. Kent died in 1973 of cancer of the liver and pancreas. Four daughters married, there are eight grandchildren. Youngest daughter is in Theology School in New York City.

Solon Abernathy and I were married in 1978, and continued in a very happy state for two years.

Presently, Ab is my activity. "Ab" is at home. He is an Alzheimer's victim.

BIOGRAPHY OF "AB"

My husband, Mr. Nice Guy.

Solon E. Abernathy was born October 11, 1908 in Pulaski, Tennessee. He was edcuated in the public schools there. He entered the University of Tennessee in 1928, and did his cooperative work in engineering with the university and Tennessee Eastman Company, Kingsport, Tennessee. In 1933, he went to work permanently with Tennessee Eastman and was with them until he resigned in 1946, to form his own business known as Abernathy-Thomas Engineering Co.

During the stay at Tennessee Eastman, Mr. Abernathy was in the Construction Shops for one year, and then was in charge of the installation of pilot plants. He then was given the job of organizing an Instrument Department the latter part of 1934. He continued as head of that department, which did the engineering, made the installations and took care of all maintenance and repair of instruments until 1942.

Mr. Abernathy was transferred by Tennessee Eastman to Holston Ordnance Works in 1942 as Project Engineer in charge of Instrumentation during the design and operation of this plant. In 1944, he went back to Tennessee Eastman as a Project Engineer.

He obtained his Tennessee Engineer's License No. 965 in 1943, and has been active in the Upper East Tennessee Chapter of Tennessee Society of Professional Engineers, having served on the Chapter Board of Directors and various committees. He was Chapter Representative to the State Board, Director on the State Board, and then Second Vice President and later First Vice President of the State Society.

Mr. Abernathy was Chairman of the Board of Abernathy-Thomas Engineering Co., with headquarters in Kingsport, Tennessee. They have offices in Knoxville and Nashville, Tennessee; Charlotte and Raleigh, North Carolina. He is Past President of the Northeast Tennessee Section of the Instrument Society of America, and was their National Delegate in 1958. He served as President of the Leslie Company, Lyndhurst, New Jersey, Sales Management Board in 1956-57. He is a member of the American Society of Lubrication Engineers and the National Association of Corrosion Engineers and Instrument Society of America. Mr. Abernathy is a member of the Kiwanis Club of Kingsport and was active in the Waverly Road Presbyterian Church.

Mr. Abernathy was much admired for his kind and generous nature and his sincere interest in people. He was a man of his word and frequently was described as a man you could trust. These attributes certainly aided him to be a success at business and a respected citizen.

Mr. GORE. Thank you. Thank you for your statement and, again, for your courage in coming and sharing it with us.

Our final witness on this first panel is Mr. Tommy Dillow, also of Kingsport, TN, whose father is suffering from Alzheimer's disease.

And, Mr. Dillow, thank you for being here, and please proceed with your statement.

Mr. DILLOW. In what I am about to say, I am expressing my own feelings as well as those of numerous other caregivers with whom I have spoken. We are offered very little in the way of treatment, no hope of cure, and sent off to care for this person.

I realized I would have to move from Atlanta where I was living at the time, knowing that the cost of professional, paid care would be more than my mother could handle. I had one choice, move home. Putting my job, lifestyle, social life, ambitions on an indefinite hold was a hard decision to make. Being single made that decision easier, but the commitment becomes increasingly more difficult to keep. Once I realized that I could be involved from 5 to 20 years, I really was overwhelmed.

At this difficult time the spouse realizes they must devote a huge chunk of their remaining life to a person who continually shrinks away from the person they once knew. Family members sometimes blame other family members for the illness, even though there is no known cause. The perceived stigma of mental illness clouds some families.

To briefly explain the reality of dealing with an Alzheimer's patient and have someone really understand is nearly impossible. Close friends and other family members don't always understand. There's always the pain of watching someone you love, hopefully, change bit by bit into someone who is less and less able to communicate even their affection for you. Those situations where there is no caring must find this an agonizing dilemma. Futilely, we try to maintain what remains of his memory by trying to keep his mind working. Anything we can do to keep him active we try, both physically and mentally.

The best analogy of the care of an Alzheimer's disease patient is that of caring for a 2-year-old child. However, this patient never improves; he only gets worse. A caretaker must dress, feed, bathe, continually check all bodily functions. This continues 24 hours a day, year after year, for as long as 20 years. They must constantly be watched so that they don't fall, or hurt themselves, or swallow something they shouldn't. The caretaker has to be cautious not to become upset over the constant mistakes the patient makes. When he goes to the bathroom on your favorite piece of furniture, it's hard to stay calm. When he chokes on a napkin while your back is turned you tend to panic. Many patients' sleep is interrupted from time to time. A caregiver finds himself going 24 hours or more without proper sleep. We must also make the inevitable trips to the bathroom during the night.

He has trouble communicating: he says he's hot when he's cold. He's always hungry even though he eats constantly; and yet, he loses weight. Knowing if and when he needs to go to the bathroom becomes more difficult. Eventually, the need for expensive equipments, or just commode chair and incontinent pants, becomes apparent. We get no assistance in paying for so many of these items we need to keep him at home.

If there is one thing I can impress on you, it is the plight of the caregiver. The patient is aware of his diminishing mental capacities, which appears to be very upsetting; but he has no real pain. If you can imagine having very few free moments, little time for anything, you come close to understanding how the caregiver suffers.

As a child I function as a secondary caregiver, a relief in an otherwise 24-hour day shift for the primary caregiver. I also take care of things Dad would if he were well. I'm there when he falls, to help him. When Mom is at wit's end, I give her a break. Dad's fierce independence keeps me from doing everything Mom can, but as he gets worse, I take on more responsibility.

So often we are not prepared to do the things we are required to do. Several times I have seen my mother stand helpless as my father turned blue, having choked because he wouldn't chew his food. I had seen the Heimlich maneuver demonstrated on television and had learned enough to save him. Because of his ravenous appetite, we must constantly prepare food which he shovels down. We've learned to prepare a special diet so he remains regular. Often it has to be chopped or cut so that he doesn't choke. He sometimes forgets to chew even while we're reminding him. As he gets worse we are learning to give enemas or suppositories. When he falls or misses a chair, he is unable to get up by himself; so we have to lift him without hurting ourselves. We have learned to physically maneuver him without hurting him or us, all the while dealing with a potentially volatile personality. His mood can change in an instant, so our words and tone must be carefully chosen and repeated often.

Keeping him occupied becomes more and more a full-time chore. As television and reading quickly become less a way to occupy time, taking him from room to room, as you try to do what you need around the house, becomes more a necessity. At times, he becomes easily confused, and that can lead to boredom and depression. Several people talking at once confuses him. This means we have little company; we are unable to be in large groups of people.

On good days, a ride in the car will ease the boredom, but getting in and out of the car becomes more and more impossible. Some patients lean to the side so badly they are unable to travel at all. Distance and physical barriers become more limited to walking. Since he always wants to be within your sight, you lose your freedom. We spend a great deal of time and energy trying to keep him happy so other things will go easier.

For instance social contacts dwindle. Those who might have sat or visited with him disappear as the situation worsens. Family caregivers are often older, and unless they are careful, this weakens them as well. Even if you can afford them, qualified paid caregivers are not easy to find.

In order to make things go smoother, we start to live at his pace. If he goes to bed early we have to as well, so we're assured a reasonably comfortable night's sleep. The nights when you're up and down all night make sure you'll stick to his schedule.

When he becomes too much for even two people to handle, I'm not sure what we will do. Although he is a veteran, the VA Hospital offers little encouragement for long-term care. Nursing homes are more and more reluctant to accept Alzheimer's disease patients because their needs are too demanding. Financial consideration is very strong. We could spend down what little they have saved over a lifetime and leave Mom with no buffer as she gets older. An otherwise healthy Alzheimer's patient falls through all the cracks in the system. Third-party payers contribute little or nothing. Even though he had an exemption, Dad volunteered to go to war; and this is the thanks he gets.

People go to great lengths to beat the system as it is now drawn, so they will qualify for Medicare insurance. It sometimes becomes almost a blessing if he has a stroke or some other illness that qualifies him for assistance. I hear stories of all sorts of very minor maladies that qualify being searched out to make an otherwise unqualified Alzheimer's disease patient eligible for paid help. Those of us who work to keep our patients healthy are further penalized. This is a definite "Catch 22" situation.

What could be done to help us? Since the illness is complex, many of the solutions may be as well. We need help in so many areas. My own personal priority would be preventative therapy. Help us use what has newly become available to try to slow the deterioration. I am successfully using choline and lecithin which I buy in the health food stores. I have requested a prescribed therapy of hydergine from our physician. There are many other projects, but information is not easy to locate. I read about such drugs as 4 amino pyridine or CI-911 and wish we could have them now. We don't have time to wait for full FDA approval for all these, we need them now. We are dealing with someone who is dying before our eyes. Many drugs would be worth a try. Anything that helps is beneficial to those of us who must maintain home care.

Education: In this locale and many others, there is a need to educate medical professionals as well as education for caregivers of a different sort. An example of this was Dad's family physician not knowing that "pre-senile dementia" is another term for Alzheimer's disease. As caregivers of newly diagnosed patients, we search rather desperately for information about the disease.

Local chapters of ADRDA are helpful where they are available, but they are short on volunteers and resources.

Respite care: A facility designed specifically to care for Alzheimer's disease patients' special needs while the caregiver allows himself to mend or keep healthy for a week or 2 each year.

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Research should take several directions:

If a way to diagnose the disease were available, a person would be able to leave their job more financially secure as a disabled person.

Research into cognitive therapy to extend and improve the quality of life.

Research into the cause and cure.

Research to learn more about how caregivers are affected.

Survey patients to focus future research so precious funds aren't wasted. So many caregivers espouse the viral or bacterial theory as a cause and a traumatic moment as the trigger for its onset. I hear this constantly.

Most importantly, quickly determine how devastating our financial needs are, and then move quickly and positively to help us.

I don't believe in throwing money at a problem, hoping it will go away. At a time when the Government needs to act more financially responsible, I feel we are way overdue for assistance. Dialysis patients get Medicare help as do cancer victims. Alzheimer's disease is certainly of equal or more severity than either of these.

Add to the sense of urgency the fact that with more people living longer we are waiting for a potential time bomb to explode. There is an indication there may be a hereditary factor which would compound the problem in a few decades. You cannot wait. You must act now to help us rid this horrible disease which not only takes the Alzheimer's disease victim but handicaps the family as well.

Thank you.

[Applause.]

Mr. GORE. Thank you very much.

[The prepared statement of Mr. Dillow follows:]

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1. If a way to diagnose the disease were available a person would be able to leave their job more financially secure as a disabled person.
2. Research into cognitive therapy to extend and improve the quality of life.
3. Research into the cause and cure.
4. Research to learn more about how caregivers are affected.
5. Survey patients to focus future research so precious funds aren't wasted. So many caregivers espouse the viral or or bacterial theory as a cause and the traumatic moment as the trigger for its onset.
6. MOST IMPORTANTLY: quickly determine how devastatin our financial needs are then move quickly and positively to help us.

I don't believe in throwing money at a problem hoping it will go away. At a time when the government needs to act more financially responsible, I feel we are way overdue for assistance. Dialysis patients get medicare help as do cancer victims. A.D. is certainly of equal or more severity than either of these.

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I am Tommy Alan Dillow.
I was born Oct. 9, 1947, in Johnson City, Tenn., adopted an only child by Mr. and Mrs. Ernest B. Dillow at my birth.
From age 5 I was raised and educated in Sullivan County, Tenn.
I attended the Univ. of Tenn.
I was honorably discharged from the U. S. Air Force.
Until 3½ years ago when my father was diagnosed as having Alzheimers' disease, I lived in Atlanta, Ga.
I now devote all my time and energy to helping my parents with no source of regular income.

Mr. **Gore**. Thank you all for very moving statements. I've participated in an awful lot of congressional hearings—not nearly as many as Senator Pepper—but I can say, from my 8 years of experience, I don't know when I've ever heard a more moving and eloquent panel in a congressional hearing, and we're very grateful for your willingness to help us in our effort to understand this horrible disease and to better outline a strategy for coping with it.

Now we'll go to questions, and I'm going to recognize Chairman Pepper to lead off the questioning.

Mr. **Pepper**. Well, I thoroughly concur with what Chairman Gore has said. I've never heard a more moving panel than you have presented here today. You have touched the heart of everyone who has heard you, with the terrible nature of this problem and the emergency that we face, and the necessity of our trying to do something about it.

Mr. Dillow, you've given a magnificently comprehensive analysis of the whole problem, what the demands are for care for these people. It's almost more than a 24-hour job for anyone to assume. And it's almost obvious that you really need two or three people to care for each person who gets in an advanced stage of this terrible disease.

And I think you've put good emphasis on the necessity for our having a conference with the Food and Drug Administration. Of course, we don't want them to loose upon the market drugs that would be harmful to people. But I remember, if I may say so, my beloved wife passed away with cancer 5 years ago, and I never shall forget that night when the doctor came and told me they had lost control of the tumor and the medicine that they'd been giving in the chemotherapy was no longer restraining the growth of the tumor. I said, "Well, have you got anything else you can possibly use?" "Yes," he said, "we've got some drugs. There may be some bad side effects from some of them." And I said, "I don't know anything else to do, so we'll just have to do the best we can."

So, if there are these people, it's just like I'm going to support on the floor of the House some legislation to authorize a doctor to give heroin to a cancer patient who is in the most grievous and terrible pain in the last days of their suffering, if they think that's the best thing to do. You've no fear then of addiction, because that poor beloved is bound for the end. So, we'll have to ask the Food and Drug Administration to look at it in the larger sense. There might be something that could be tried that would be reasonable in its expectations of doing good, maybe, that they could help.

Chairman Gore has just called my attention to the fact that there are 400,000 veterans over 65 years of age with Alzheimer's disease. Now, if anybody deserves the gratitude of their country, it's those people who offered their lives for their country's service. I can't believe that our Government can go on—we are a great Nation, a great people, and we are grateful people; I can't believe that we could go on giving no aid to those terrible people. There are very few families that are fortunate enough to have a son like you that will give your whole life to the care of a beloved one. Many of those veterans don't have people like that to help them.

So, the gist of it is, as I see it from what you've said, something like Mr. Gore and I are proposing—it seems in the right direction—to increase our research facilities, to get those facilities out among the people in the country, where they can be in close contact with these patients, and their families, and their loved ones, and, at the same time, to try to provide assistance to the families in providing the care that they have to have some way or another.

You know, when President Kennedy said, a good many years ago, we were going to the Moon, he didn't say, "Provided that we can go there for a billion dollars"; he said, "We're going to the Moon." And when they started out to make the nuclear bomb, during the war, they didn't say, "We're going to build it down here in Tennessee provided we can build it cheap enough"; they said, "We're going to build the bomb and end the war with it, that's our intention." So I think we've simply, with our characteristic national courage, and fortitude, and determination, have got to come to grips with these terrible killers that are devastating our people. If an enemy were coming into our country and taking several hundred thousand a year of our people away, why we'd spend every cent that we have defending them, protecting them; and now, these people who are so helpless but they cry out to our sympathy with the tragedy of their illness.

So, it seems to me you all have surveyed the picture here very thoroughly this morning, and I think you've given great impetus, maybe, to our colleagues in the Congress supporting us in this effort to try to do something to help those wonderful people who are beset by this terrible disease. So, if you wish to add anything more to what you've said, either one of you——

[Negative response.]

Mr. Pepper. Well, thank you very much.

[Applause.]

Mr. Gore. Thank you, Senator Pepper.

Let me ask a couple of brief questions, if I may.

Each one of you has described the enormous amount of time required of family members to care for Alzheimer's victims. Doesn't it make sense to look at respite care centers? And what I mean by that is——

Well, let me back up. Two days ago I visited a center in Memphis that only cares for 10 people, because they get voluntary contributions and it's run by volunteers. But they offer a service for Alzheimer's families whereby the family can take a victim of Alzheimer's Disease to this center for 6 hours a day, 2 days a week. During that time, they can get shopping done, they can have just a short break and collect themselves and get a little bit of rest and do the various chores that need to be done. And during those 6 hours, two days a week, the victims are given exercise so they burn off some of their high metabolism and they're given what they refer to as reality orientations. They sit down and tell them, "This is what day it is, it's summer"—and you all are nodding and smiling because you do that, I know, frequently, every day.

But if we wanted to select one thing in addition to the research needs and in addition to getting the medical communities to be more forthcoming and more aware of what families need when they're confronted with these circumstances, would that be quite a high priority?

Dr. Summerlin, you as a professional and as a family member, what is your reaction to that suggestion?

Dr. SUMMERLIN. I think that would probably be helpful to some families and in some situations. I'm not sure that it would be the answer in all cases. Speaking again from my own perspective, since I'm the only one that has a car and is the provider of the transportation, and I do have a full-time job, it would be extremely difficult for me in this instance to get my family member to the respite care—get my mother, do all the chores that you're suggesting——

Mr. GORE. Right.

Dr. SUMMERLIN [continuing]. The banking and shopping and all of that. So, there are other needs that are associated with that, such as transportation, you know, and some of the other kinds of aspects. It is a form of help, yes.

Mr. GORE. It would just depend upon the circumstances——

Dr. SUMMERLIN. Yes.

Mr. GORE [continuing]. That each family finds itself in.

Mrs. ABERNATHY. May I reply?

Mr. GORE. Please do.

Mrs. ABERNATHY. I'm like my friend here. It would be too difficult to get my husband into the car. If I did get him in the car, if I could, he would open the door on the highway. It would take several people to supervise that ride to get him there, the bath before we got there, the plastic pants, et cetera.

Mr. GORE. OK.

Mrs. ABERNATHY. So, it would not help me.

I think, early stages, with their still being aware, they might resent it. Their pride might be insulted. I think you have to consider that, that at times they are lucid. Almost to the end some bright spot shows up. So, I think that's a consideration.

I'd like to suggest that a care center might be, as they do in England where the family does care for this person, but 30 days out of the year grandmother, mother, my husband, would be brought to the center. You are given 30 days off. The children, the grandchildren, don't have to be bombarded by mama's million questions, et cetera. And those 30 days are spent with intensely examining the patient, monitoring the patient, new passive exercises she or he might need. When the 30 days are up, and the relatives come to pick up this person, you've got a whole program designed for you. The victim has had a month off, the family's had a month off. I'd find that appealing, interesting to check out.

Mr. GORE. It would be less than 1 day a week off from 24-hour-a-day care. That's interesting.

Mr. Dillow.

Mr. DILLOW. As I said in my previous remarks, the problem is complex and I think you will find the solution will be complex. It's very, very difficult to explain all the problems involved. This would be a good idea for some people. In our particular situation at home, we're very special. My dad was diagnosed 3½ years ago. He's still relatively mobile. He has good days, he has bad days. He has days when he could get in and out of a car, he has days when you couldn't get him in and out of a car if you tried.

In my experience with talking with other people, I am an exception. I think Ab, Bea's husband, was diagnosed in roughly the same period of time; he is immobile. There is no way for him. In the cases that I have talked to, generally after a year, possibly a year and a half, they become immobile. It would help some people in the early stages, in that they would not get dragged down so far so quickly. In other words, it would postpone the inevitable. If you have a year or two at the beginning of this that it doesn't tackle you quite so hard, that would be of benefit. But it's going to take, probably, a number of solutions to help all stages of this disease.

It's just like I said, it will last for 5 to 20 years. The patient changes constantly during that period of time. One answer is not going to fit all people.

Mr. GORE. OK. Well, that's the reason why we ask questions, to get answers and learn from them; and I appreciate that. We're going to try to take a comprehensive view during this day of hearing and the other 2 days, and try to have some comprehensive recommendations about all of these strategies.

Recognizing that we are at the very beginning of this hearing, let me just ask you another question. And I ask it because there are so many people here with us whose lives have been touched by this illness.

Based upon your experience, and recognizing the fact that you've had very little assistance from the medical professions as a whole because we're really just, as a country, becoming aware of this—it's been confused with senility for so long that we haven't focused on it—but recognizing our limitations and the lack of cumulative experience, if you had a chance to talk to a family member whose parent or spouse had just gotten the diagnosis of Alzheimer's disease, and you could give them some kind of advice, what would you tell them about to help them with what lies ahead? Understanding that we're going to try to come up with some recommendations for the whole country, to answer that question, but just based on your personal experience, what would you tell them?

Mr. DILLOW. The first thing I would tell them is, it's not simple. Bea and I are involved in organizing an Alzheimer's support group in this area. That is exactly the function of that group. It's such a complex disease. I'm 3½ years into this and I continually learn more about it—learn little tricks to make care easier, all sorts of things. What our intention is to do, if we can get the resources to do it, is to have a packet of information that we can hand to these people and let them learn. You cannot sit down with a—that's one of the most frustrating times with an Alzheimer's family, is to try to sit down with them and explain to them: One, what the disease is; two, what they can expect. It's very, very difficult, in any short period of time, to explain all that to them. That's one of the biggest problems with it, you cannot just sit down and say "this is what's going to happen."

Mr. GORE. Yes.

Mr. DILLOW. And each different case is different, each personality is different. I know of cases where the personalities are very volatile, where it's almost impossible to handle the patient. In my case, my dad's a gentle person. He reacts a different way. There are all sorts of nuances of different personalities that affect it. It's just one of those things that you have to take on an individual basis.

Mr. Gore. We're going to hear more about support groups later on in the hearing, and we will give information to people here interested in contacting the support groups on how to do so. In addition we will hear, from our third panel, a very excellent statement from Eleanor Carper in Memphis about a study done that shows 73 percent of all of the families of Alzheimer's victims report that they have received virtually no guidance or help from the medical community when the diagnosis first occurs.

And of course, again, the medical community, like all the rest of us, is just becoming aware of what this really involves, and hopefully that will change. We're going to have some recommendations there, too.

The same question, Mrs. Abernathy.

Mrs. Abernathy. We were fortunate that after two doctors, we were sent to Duke for a diagnosis, and their center did prepare me better than any physician's office had—made suggestions and, rather, projected what our life would be like, that we needed 24-hour help in the house immediately, you know, this sort of thing, and that helped.

If, in this area, rather than having to go to North Carolina or go to Duke—which was wonderful to us—but if we could establish a center in this area where families could be referred to, could be educated, the physicians could be educated, I think it would help tremendously, rather than have the patient diagnosed "he's Alzheimer's, I really can't help you, goodbye," a center that could go on supporting the different stages.

Mr. Gore. Well, we're going to work on precisely that.

Dr. Summerlin.

Dr. Summerlin. One of my problems is that once the diagnosis is made, it's almost—all of the pain, and all of the emotion, and all of the trying to figure out what's going on with the family member is almost accomplished; not always, in every case, but at least in our case it was. And the kind of support that one needs really is before the diagnosis, not necessarily after the diagnosis is made; because by then you have had some information about the disease, you have some idea of where you are with it, where you're going with it.

The kinds of support that I would recommend to the individual at that time is to look for financial resources to help with the problem, look for legal counsel with the legal problems; you know, find transportation, find volunteers, find anything and everything you possibly can as well as resources to help you get through this. And that is not easy to do.

Mr. Pepper. Mr. Chairman, may I just say one word?

Mr. Gore. Surely.

Mr. Pepper. You remember Dr. Butler, who was head of the Aging Institute in Washington for many years, did a marvelous job in the service of the elderly of our Nation. He was the first one that I ever heard of who said that this Alzheimer's disease was not senility, it was a disease. It was generally being considered that it was a decline, that when you got old you got senile, you got this what they then called senility. And he has been the leader in advocating that the medical schools of the country put in their curriculum geriatrics, the problems of the elderly. A lot of the doctors that come out of some of the fine medical schools don't know anything much about the diseases of the elderly, and their symptoms are not always the same—the reaction to drugs and treatment, as you ladies know, is not always the same. So, one of the things that we'll have to include in our program is to encourage a study in the medical schools of the particular problems of the elderly by the young doctors, so when they come out they'll know what they're dealing with.

Dr. SUMMERLIN. I'd like to expand that to nursing schools as well.

Mr. PEPPER. Well, of course, yes, I'm sure you're exactly right. Because I consider the nurses about equal to the doctor——

Dr. SUMMERLIN. Thank you.

Mr. PEPPER [continuing]. In treatment of patients.

[Applause.]

Mr. GORE. And you can rest assured, Senator Pepper, that that approach is now being taken here at East Tennessee State.

We could spend a great deal more time with this panel, but we have only a short amount of time for the entire hearing, so we're going to have to move on to the next panel. But I want to, again, thank you very much for your grace and courage in appearing here and helping us with our effort to understand and cope with this matter. Thank you very, very much.

[Applause.]

Later in the hearing I will have a statement about other information on how family members and others interested in Alzheimer's disease can get information about how to cope with it and how to deal with it.

Now, our second panel is made up of two of the leading experts in the entire country on this matter: From the National Institute on Aging, Dr. Zaven Khachaturian, if you would come forward to the witness table; and from the Office of Technology Assessment in Washington, Dr. Robert Cook-Deegan.

And while you all are coming forward to the witness table—yes, please come and join us—while you are coming forward to the witness table, let me make an announcement that I neglected or forgot to make at the beginning of the hearing: This hearing is going to end at between 12:45 and 1 o'clock, and then we're going to get some lunch. And those here who would like to meet Senator Claude Pepper, there is a reception after the hearing in rooms 6 and 7 at 2 o'clock. And you may want to get some lunch after the hearing and then come back to the reception. You may suit yourselves, but he will be here for a reception at 2 o'clock at rooms 6 and 7. It's right across the hall from this room at 2 o'clock.

Now, to our second panel who will give us an overview of the technical and medical issues, we have asked all of our witnesses to keep their statements relatively short, so as to save time for questions and because we have a long hearing in a short time period. Without objection your entire prepared statements will appear in the record in full, and we'd like to invite you to begin with your presentation at this time, beginning with Dr. Zaven Khachaturian from the National Institute on Aging.

Thank you for being here, and thank you for making your long journey to help us here today. And please proceed.

STATEMENT OF ZAVEN KHACHATURIAN, PH.D., CHIEF, PHYSIOLOGY OF AGING BRANCH, NATIONAL INSTITUTE ON AGING, NATIONAL INSTITUTES OF HEALTH, ACCOMPANIED BY ROBERT M. COOK-DEEGAN, M.D., SENIOR ANALYST, BIOLOGICAL APPLICATIONS PROGRAM, OFFICE OF TECHNOLOGY, U.S. CONGRESS

Dr. Khachaturian. Thank you, Mr. Chairman, for having me here at this hearing. Mr. Chairman, I speak as a representative of the National Institute on Aging, the Federal agency with the primary responsibility for conducting supporting research on biomedical and behavioral processes of aging and the diseases of the aging.

Since its inception in 1974, the institute has targeted Alzheimer's disease as one of its most important research priorities and challenges. Within the Public Health Service of the Department of Health and Human Services, the NIA shares an interest in responsibility for this disease with its sister institutes at NIH.

During the past 7 years, I have been responsible for developing and administering NIA's program of research on Alzheimer's disease. During that period I have been in a position to see the growth of research on this devastating disease from very modest beginnings to a responsible national scientific effort. The things I have to say may appear trivial in the face of the very moving testimony I've just heard. What I have to say does not have any immediacy or the quick solutions to this very complex problem.

Of the many brain impairments in the aged, decreased cognitive functioning is generally accepted as the more serious problem. The NIA's interest in Alzheimer's disease stems from the fact that this is the most severe form of cognitive impairment in old age. We know that what we call Alzheimer's is a disease and not part of the normal aging. We don't know yet whether the brain changes associated with Alzheimer's disease represent an accelerated form of normal aging. Scientific evidence suggests that many of the structural and biochemical changes in the aged brain differ quantitatively, not qualitatively, from those in dementing disorders such as Alzheimer's disease. As a result, it may be difficult to distinguish between normal brain aging and disease. We hope that studies of changes in brain and their relation to memory decline will help throw light on this issue.

In the next several minutes, I will attempt to provide an overview of the magnitude of the problem presented by Alzheimer's disease and the remarkable scientific achievements that have already been made.

The number and the proportion of elderly in the United States are rapidly increasing and are already having profound influences on American economic, social, and political thinking and behavior. Biomedical research and its applied technologies have made it possible for an increasing number of people to survive until their seventies and eighties. Perhaps the most significant impact will prove to be economic—the costs of needed health care. Persons 65 and older are at greatest risk for disease and, consequently, tend to be—have the greatest need for medical services. Many age-related disabilities tend to be chronic rather than acute, requiring long-term care. Such care is expensive, and costs are expected to rise at an exponential rate due to the increasing number of the elderly.

Among the many age-related disabilities, those related to changes in the central nervous system or brain functioning have important implications for both public policy relating to health services and national priorities for biomedical research. This is true because failure in cognitive functioning is one of the principal indicators for institutionalization of the elderly in long-term care facilities. Long-term custodial care in an institutional setting is extremely expensive, often impersonal, and may contribute to further deterioration of health.

Approximately 10 percent of the elderly population show clinically significant cognitive deficits. The frequency of intellectual impairment is considerably higher among those living in nursing homes, where an estimated 50 percent or more have some form of mental impairment. A significant number of these are now recognized to have Alzheimer's disease. In 1982, the national annual expenditure for nursing home care for the aged was approximately $27 billion. During the next 10 years it was estimated that this will become a staggering amount of $80 billion in constant dollars.

An important issue is that of accurate diagnosis. It is estimated that up to 20 percent of persons who have been labeled "demented" may have reversible brain disorders that require evaluation and treatment. Misdiagnosis is extremely costly and a tragic waste when it leads to long-term institutionalization. There is clear and urgent need to continue the quest for scientific knowledge concerning diagnosis and treatment possibilities for dementia and the biological mechanisms both of aging and such dementing disorders of Alzheimer's disease.

The dimensions of the health problems posed by age-related changes in the central nervous system are vast. Basic research into the neurobiology of aging as the route to resolving this problem has been demonstrably successful in advancing knowledge of the human brain and providing the first real insight into some of the more baffling neurological disorders. The treatment of Parkinson's disease patients with L-dopa to replace loss of vital brain chemicals and the discovery of enkephalin and endorphin, the brain's own "opiate" drugs, are important examples.

These advances have come about because of a critical combination of talent and technology in neuroscience. It is now possible to obtain a detailed picture of what happens at the molecular level of nerve cells as well as a clearer idea of how large clusters of nerve cells and systems operate in elaborating complex human behaviors. Enthusiasm in the field of neuroscience is running high, and researchers are optimistic about the future of brain research.

Although systematic studies of age-related changes in the human brain have a 100-year history, strong interest in this subject is recent. During the last decade, a growing community of neuroscientists, spurred partly by the availability of Federal funds for research, has begun to study a broad range of topics in the neurobiology of aging. These range from basic molecular to clinical issues, and from the physical chemistry of membrane transport mechanisms to the neuropsychiatric assessment of the aging brain. Not long ago little was known about senility and Alzheimer's disease was considered a hopeless affliction of the aged. Now we know that chemical and physical changes in the brain can account for some of the memory deficits in Alzheimer's disease and benign forgetfulness of the aged. The remarkable pace of scientific progress in this area of research increases the likelihood that some forms of dementia may be treatable by pharmacological agents.

Scientific research has also made it possible to start formulating researchable theories about the pathogenesis of Alzheimer's disease and to begin assembling the pieces of the puzzle of what causes the disease.

There is considerable evidence that the complex and delicate processes of cell-to-cell communication are affected in the aging brain. In the case of Alzheimer's disease, there is compelling evidence that at least one neurotransmitter system—that system involving the neurotransmitter acetylcholine—is affected. It is now well established that there is a significant decrease in the activity of choline acetyltransferase, a key enzyme in acetylcholine synthesis.

The suggestion that observed changes in the enzyme CAT—which is the short for choline acetyltransferase—may actually be due to brain cell death was recently confirmed by a group of investigators at Johns Hopkins University, who discovered that Alzheimer patients have severe losses of neurons in part of the brain called nucleus basalis of Meynert. Most of the neurons in this nucleus send their axons directly into the cortex, the outermost layer of the brain, and provide the major source of cortical cholinergic innervation. The findings of this study will enable scientists to ask such specific questions of why neurons in the nucleus basalis have specific vulnerability, and what events trigger the degenerative processes.

The other common biochemical abnormality that occurs in dementing disorders is a decrease in the cerebral metabolic rate. Cerebral blood flow is regulated to meet the requirements of the cerebral metabolism and functioning. Glucose is the major substrate for oxidative metabolism in the brain. A number of studies in dementia patients have shown decreases in cerebral blood flow, oxygen consumption, and cerebral glucose utilization.

This finding that cerebral glucose utilization is decreased in Alzheimer's disease is an important scientific observation because the synthesis of acetylcholine is strongly dependent on carbohydrate metabolism. Consequently, it appears that the neurotransmitter deficit observed in Alzheimer's disease may be due to a fundamental problem in glucose utilization or energy metabolism in the nerve cell.

Of the major observable physical abnormalities in the brains of Alzheimer's patients, neurofibrillary tangles amd senile plaques are the two most characteristic lesions. The concentration of these lesions is correlated both with the severity of dementia and with deficiencies in the cholinergic system. The lesion can occur in the hippocampus of most aged individuals without dementia and their frequency appears to increase with age.

In Alzheimer's disease, however, neurofibrillary tangles are present in large numbers. Attempts to characterize the biochemical composition of neurofibrillary tangles have not yet succeeded. We do know, however, that neurofibers that make up the tangles consist of a pair of filaments wound around each other forming a paired helical filament, and that these filaments bind together in a rigid, highly insoluble bond in a way that seriously threatens the life of the cell.

All of these results are extremely important because they provide a crucial beginning for piecing together the Alzheimer's disease puzzle. It is now possible to formulate testable hypotheses that can provide a common framework for interrelating a number of the discrete results discussed here. For example, in study after study, we are seeing the importance of calcium levels within the cell in terms of cell-to-cell communication and even the viability of the cell itself. Time after time, findings in Alzheimer brains point to a disruption in the delicately balanced homeostatic system of transporting calcium across the cell membrane. Such a disruption of the system could be a consequence of exposure to a neurotoxin, such as aluminum; pathogenic agents, involving either genes or infectious agents; other insults; or even aging. Further study should allow us to develop integrated theories of aging and better understanding of the biological mechanisms of diseases such as Alzheimer's disease.

In summary, the systematic study of Alzheimer's disease began only within the last decade, despite the fact that the disease was first described nearly three-quarters of a century ago. In this brief period of time, our knowledge about this disease has advanced from nearly total ignorance to a level where it is possible to begin testing possible treatments and formulating viable hypotheses to explain its causes.

The scientific community now has the tools, the knowledge base, and the means to solve the problem of Alzheimer's disease within the next decade. This remarkable progress in dementia research has been made because of the availability of scientific knowledge, technology, and methods; that is, the bricks and mortar to start building a structure. This has been more than equaled by increasing interest and enthusiasm in the medical community and among the general public.

The Public Health Service, of which the National Institute on Aging is a part, has supported a significant proportion of the basic biomedical research in this country during the last 30 years. A tremendous amount of this work has had direct and indirect bearing on our understanding of the basic mechanisms of cell function, including neurons. Using knowledge gained from such areas of science as biochemistry, cellular and molecular biology, bioengineering, physics, genetics, recombinant DNA, and cloning technology, it is now feasible to make rapid progress in understanding the cause or causes of Alzheimer's disease as well as other disabling disorders of the aged.

Thank you for this opportunity to speak with you about Alzheimer's disease.

[Applause.]

[The prepared statement of Dr. Khachaturian follows:]

STATEMENT BY

Zaven Khachaturian, Ph.D.
Chief, Physiology of Aging Branch
National Institute on Aging
National Institutes of Health

Mr. Chairman, I speak as a representative of the National Institute on Aging (NIA), the Federal agency with the prime responsibility for conducting and supporting research on the biomedical and behavioral processes of aging and the diseases of aging. Since its inception in 1974, the Institute has targeted Alzheimer's disease as one of its most important research priorities and challenges. Within the U.S. Public Health Service of the Department of Health and Human Services, the NIA shares an interest and responsibility for this research with its sister Institutes: The National Institute of Neurological and Communicative Disorders and Stroke, The National Institute of Mental Health, and The National Institute of Allergy and Infectious Diseases.

Of the many brain impairments in the aged, decreased cognitive functioning is generally accepted as the most serious problem. The NIA is interested in Alzheimer's disease because it is the most severe form of cognitive impairment in old age. Although we consider Alzheimer's a disease of the aged, we have not yet resolved the scientific issue of whether brain changes associated with this disease and other forms of dementia represent an accelerated form of normal aging.

Evidence suggests that many of the structural and biochemical changes in the aged brain differ quantitatively, not qualitatively, from those in dementing disorders such as Alzheimer's disease. As a result, it may be difficult to distinguish between normal brain aging and disease. We hope that studies of changes in the brain and their relation to memory decline, for example, will help elucidate this issue.

In the next several minutes I will attempt to provide an overview of the magnitude of the problem presented by Alzheimer's disease, and the remarkable scientific achievements that have already been made.

The number and proportion of elderly in the United States are rapidly increasing, and are already having profound influences on American economic, social, and political thinking and behavior. Biomedical research and its applied technologies have made it possible for an increasing number of people to survive until their seventies and eighties. Perhaps the most significant impact will prove to be economic--the costs of needed health care, clinical medicine, and biomedical research. Persons 65 and older are at greatest risk for disease and, consequently, have the greatest need for medical services. Many age-related disabilities tend to be chronic rather than acute, requiring long-term care. Such care is expensive, and the costs are expected to rise at an exponential rate due to increased numbers of elderly.

Let's review some of the most dramatic demographic changes of this century:

- The number of people over 65 has risen from 3 million in 1900 to 27 million today and is projected to reach 43 million by 2020--nearly double the current total. At the same time, the proportion of those over 65 has risen from 4.1 percent in 1900 to 11 percent in 1980.

- The greatest increases are among the "old-old," people who are likely to require special care. The segment of the population over age 85 is expected to grow rapidly over the next 40 years, and to reach 5 million by 2020.

- Although nearly 68 percent of older people consider themselves healthy and 90 percent live independently, nearly 86 percent have one or more chronic health conditions or disabilities.

- The elderly account for 15 percent of all visits to physicians, 34 percent of all days spent in short-stay hospitals, 89 percent of all nursing home occupancy, and 29 percent of all money spent on personal health care. In 1984 the total cost of health care services to the elderly is expected to reach $120 billion.

Among the many age-related disabilities, those related to changes in the central nervous system (CNS) or brain functioning have important implications for both public policy relating to health care services and national priorities for biomedical research. Age-related changes in brain functioning tend to accompany loss in physical ability. Impaired hearing, vision, motor skills, and cognitive functioning profoundly affect the quality of life for the aged. Such changes frequently influence the attitude of others towards the aged, affect the individual's self-image, and can determine the nature and quality of health care services. The elderly person with cancer or heart disease often evokes feelings of sympathy; one with a hearing problem is likely to elicit feelings of impatience and be perceived as "difficult." An elderly person with failing memory is labeled senile; in fact, failure in cognitive functioning is one of the principal indicators for institutionalization of the elderly in long-term care facilities. Conversely, an older person with a serious physical disability may not be institutionalized because family members find it more tolerable to care for a person with physical infirmities. Long-term custodial care in an institutional setting is extremely expensive, often impersonal, and may contribute to further deterioration of health.

Approximately 10 percent of the elderly population show clinically significant cognitive deficits. The frequency of intellectual impairment is considerably higher among those living in nursing homes, where an estimated 50 percent or more have some form of mental impairment. A significant number of these are now recognized to have Alzheimer's disease. In 1982, the annual national expenditure for nursing home care for the aged was approximately $27 billion (Health Care Financing Review, Fall 1983). During the next ten years it is estimated that this expenditure will become a staggering $80 billion in constant dollars.

Yet another important issue is that of accurate diagnosis. There are as yet no specific studies identifying the frequency and cause of reversible cognitive impairments in the elderly, but it is estimated that up to 20 percent of persons 65 and over who have been labeled "demented" may have reversible brain disorders that require evaluation and treatment. Misdiagnosis is extremely costly and a tragic waste when it leads to long-term institutionalization. There is clearly an urgent need to continue the quest for scientific knowledge concerning the diagnosis and treatment possibilities for dementia, and the biological mechanisms of aging and such dementing disorders as Alzheimer's disease.

The dimensions of the health problems posed by age-related changes in the central nervous system are vast. Basic research into the neurobiology of aging as a route to resolving these problems has been demonstrably successful in advancing knowledge of the human brain and providing the first real insights into some of the more baffling neurological disorders. The treatment of

Parkinson's disease patients with L-dopa to replace the loss of a vital brain chemical, dopamine, and the discovery of the enkephalins and endorphins--the brain's own "opiate" drugs-- are important examples.

These advances have come about because of a critical combination of talent and technology in neuroscience. It is now possible to obtain a detailed picture of what happens at the molecular level of a nerve cell as well as a clearer idea of how large clusters of nerve cells and systems operate in elaborating complex human behaviors. Enthusiasm in the field of neuroscience is running high, and researchers are optimistic about the future of brain research. In several areas of investigation, convergent approaches are focusing upon key issues of neurobiology.

In order to move toward the fundamental goal of prevention of nervous system disorders, develop effective treatments for those presently afflicted, and ensure the rapid transfer of research advances to clinical applications, it is essential to capitalize on this new momentum in neuroscience.

Progress in biological science needs good ideas, appropriate biological models--and the necessary technology--the methods and tools to make the observations fruitful.

The neurosciences can now command all three of these elements. There has never been a lack of good ideas or experimental models, but only in the last 20 or 30 years have there been significant gains in technological prowess. These achievements in technology are expected to continue making a substantial difference in the rate of advancement of neuroscientific knowledge.

Although systematic studies of age-related changes in the human brain have a 100-year history, strong interest in this subject is recent. During the last decade a growing community of neuroscientists, spurred partly by the availability of Federal funds for research, has begun to study a broad range of topics in the neurobiology of aging. These range from basic molecular to clinical issues, and from the physical chemistry of membrane transport mechanisms to the neuropsychiatric assessment of the aging brain. Not long ago, little was known about "senility," and Alzheimer's disease was considered a hopeless affliction of the aged. Now we know that chemical and physical changes in the brain can account for some of the memory deficits in Alzheimer's disease and benign forgetfulness of the aged. The remarkable pace of scientific progress in this area of research increases the likelihood that some forms of dementia may be treatable by pharmacological agents.

Research supported by the U.S. Public Health Service has also made it possible to start formulating a coherent scientific story on the pathogenesis of Alzheimer's disease, and to identify a number of exciting scientific opportunities that are ready to be exploited. Of even greater importance is that it is now possible to propose testable hypotheses that can provide a framework for interrelating some of the observations reported by scientists working in this area of research.

The cell-to-cell communication system in the brain is very complex and delicately balanced. Its proper functioning relies on a number of interdependent processes. The disruption of any one of these can have a deleterious influence on the whole system--injury, infection, disease, or aging are among the factors that can affect this vital communication system in a number of ways. For example, changes in the cell's excitability, ability to synthesize, transport, or release adequate amounts of neurotransmitter, or changes in the sensitivity of receptors can influence whether or not a cell will transmit the messages it receives.

There is now considerable evidence that it is precisely these processes of cell-to-cell communication that are affected in the aging brain. In the case of Alzheimer's disease, there is compelling evidence that at least one neurotransmitter system is affected.

Two of the main biochemical abnormalities that occur in aging and dementing disorders, especially in Alzheimer disease, appear to involve defects in neurotransmitter synthesis and cerebral metabolism. Although the precise relationship between physiological abnormalities in the brain and memory failure in Alzheimer disease is complex, there is considerable evidence to suggest that one of the most common biochemical disorders in dementia occurs in the neurotransmitter systems. In recent years the cholinergic system (neurons using the neurotransmitter acetylcholine for communication) has attracted the most attention. It is now well established that there is a significant decrease in the activities of choline acetyltransferase (CAT), a key enzyme in acetylcholine synthesis, in aged and Alzheimer brains. A reduction in the activity of such important enzymes in the brain may lead to reduced production either of neurotransmitters or of key intermediary ingredients necessary for their synthesis. Although the focus is on the cholinergic system in aging and dementia, there is some question as to whether this is the only transmitter system to undergo dramatic changes. Several studies have shown that age-related changes occur in the synthesis and release of other neurotransmitters, such as catecholamines.

The suggestion that observed changes in the enzyme CAT may actually be due to brain cell death was recently confirmed by a group of investigators at the Johns Hopkins University, who discovered that Alzheimer patients have severe loss of neurons in a part of the brain called the nucleus basalis of Meynert.

Most of the neurons in this nucleus send their axons directly into the cortex (the outermost layer of the brain) and provide the major source of cortical cholinergic innervation. Previous studies on autopsy material from Alzheimer patients had demonstrated a reduction in markers for acetylcholine-using neurons in those parts of the brain involved in learning and memory, such as the hippocampus and cortex. The results of the Hopkins study are extremely significant because they show for the first time that cholinergic neurons in the nucleus basalis of Meynert may selectively degenerate in Alzheimer disease. Establishing a relationship between the loss of neurotransmitter-specific cell population and a major disorder of memory functioning such as Alzheimer's disease is an important scientific achievement. The findings of this study will enable scientists to ask such specific questions as why the neurons in the nucleus basalis have specific vulnerability, and what events trigger the degenerative process.

In a related study using animals, a group of investigators at the University of California, Irvine, has shown that a serious metabolic disorder occurs in nerve cells, particularly in those parts of the brain concerned with memory function, when the hippocampus, the major input to this part of the brain, is surgically eliminated. This study is important because it provides an experimental basis for explaining the clinical observations of the Hopkins scientists. The Irvine animal studies show that eliminating the major input to the hippocampus significantly reduces the transport of free calcium ions (Ca^{2+}) into the mitochondria of neurons in the hippocampus. The mitochondria are important organelles inside the cell which are involved with the synthesis of several crucial enzymes such as pyruvate dehydrogenase. The decreased ability of the mitochondria to take up the free Ca^{2+} from inside the cell may have important functional consequences for cells that have their inputs eliminated. The reduced capacity of a nerve cell to buffer or regulate the concentrations of free calcium inside the cell may cause the concentration of calcium to rise, with serious consequences that eventually may lead to cell death.

Among the major scientific issues that have not yet been resolved are how nerve cells recognize that their input has been eliminated, and what factor or factors responsible for initiating the process of specific cell loss in aging and dementing disorders. It has been suggested that the cell loss in the nucleus basalis of Meynert in Alzheimer patients may reflect an abnormality of a neuromodulator in the brain that is specific to cholinergic neurons. Neuromodulators, a class of chemicals found in the brain, are believed to modify the excitability of neurons. The presence of a neuromodulator at a synapse may enable a neuron to be more reactive to certain neurotransmitters; conversely, the presence of other neuromodulators may reduce the reactivity of a neuron. In this manner, neuromodulators influence the probability of whether or not certain types of information will be transmitted by a neuron.

Only a few of the numerous potential modulators of cholinergic neuronal function have been studied to date. But we know that complex interactions exist between various neurotransmitters, and that a given neurotransmitter may act as a modulator of another neurotransmitter.

Dr. Stanley Appel, of Baylor Medical Center, has suggested that the presence of an extrinsic factor, neurotrophic hormones, influences the maintenance and survival of neurons. A reduction or blockage in the release of neurotrophic hormones could lead to cell degeneration. According to this theory, the selective vulnerability of cholinergic neurons in Alzheimer's could be due to a defect in the neuromodulator system or a neurotrophic hormone. Research in this important area is under way at several universities.

The other common biochemical abnormality that occurs in dementing disorders is a decrease in the cerebral metabolic rate. Cerebral blood flow is regulated to meet the requirements of cerebral metabolism and function. Glucose is the major substrate for oxidative metabolism in the brain. A number of studies in dementia patients have shown decreases in cerebral blood flow, oxygen consumption, and cerebral glucose utilization.

The finding that cerebral glucose utilization is decreased in Alzheimer's disease is an important scientific observation because the synthesis of acetylcholine is strongly dependent on carbohydrate metabolism. Any reduction in oxidative metabolism or glucose utilization should therefore lead to reduced acetylcholine synthesis. Gary Gibson, of Cornell Medical School, has shown that cholinergic neurons may be more susceptible to impaired carbohydrate metabolism than any other neurotransmitter systems. Consequently, it now appears that the neurotransmitter deficit observed in Alzheimer's disease may be due to a fundamental problem in glucose utilization or energy metabolism of the nerve cell.

The enzyme pyruvate dehydrogenase complex (PDHC) occupies a pivotal position in regulating energy metabolism. In the brain, enzyme PDHC relies on glucose as the main source of energy. A group of British scientists has suggested that PDHC activity may determine the amount of acety-CoA, an essential constituent of acetycholine, available for the synthesis of neurotransmitter. Decreases in the activity of this enzyme may thus exacerbate the cholinergic deficiencies of Alzheimer patients. Investigators at Cornell Medical Center have in fact found a significant decrease in the amount of the enzyme PDHC measured in autopsy samples of Alzheimer brains. The low concentration of PDHC in these brains may be pathophysiologically significant because, as indicated earlier, reduced metabolic rate and cholinergic dysfunction are two well-defined characteristics of Alzheimer's disease.

The enzyme PDHC plays a central role in the cholinergic memory disorder of Alzheimer's disease. Acetyl-CoA (an ingredient of acetycholine) is generated in the mitochondria through PDHC. There is evidence that PDHC activity is closely related to the speed with which mitochondria perform their critical functions of picking up or sequestering the excess free calcium ions inside the neuron. Hence, it appears that PDHC activity is related to regulating the concentration of free calcium ions which, if allowed to rise, can lead to cell damage. This suggests that a disturbance in the activity of the enzyme PDHC could result in not only a neurotransmitter deficiency but in neuronal death.

Increasing the net concentration of free calcium ions (Ca^{2+}) within the neuron beyond tolerable limits could have a number of serious consequences because of the key role Ca^{2+} plays as a second messenger within the cell. Second messengers such as Ca^{2+} and cAMP play a key role within the cell communication system by regulating a number of biochemical activities within the cell. One of the most important effects of increasing Ca^{2+} concentrations is on the axonal transport. Axonal transport, as a two-way transportation system, plays a vital role in the normal functioning of a neuron. Inside the cell body of a neuron, functionally important substances are synthesized under the control of the cell nucleus and are transported down the axon to its terminals, where neurotransmitters are stored inside vesicles. The normal functioning of axonal transport depends on many factors, such as axonal continuity, cell suma integrity, oxidative metabolism, microtubules, neurofilaments, and Ca^{2+} concentration.

A blocked axonal transport, regardless of its origin, could prevent materials that are essential for neurotransmission from reaching nerve terminals, and could interfere with other vital functions, resulting in nerve death. Recent experiments have shown that there is a calcium-activated enzyme called protease within the neuron. This chemical, which specializes in degrading proteins such as neurofilaments, may be involved in the normal degradation (cleaning-up) of the neurofilaments' proteins when they reach the axon terminal. Since protease is activated by the presence of calcium, any alterations in calcium concentration or changes in protease activity could lead to abnormal accumulation of neurofilaments in the axon terminal.

The major observable physical abnormalities found in the brains of Alzheimer patients include granulovacuolar degeneration, Hirano bodies, neuronal loss, neuritic or senile plaques, and neurofibrillary tangles. Neurofibrillary tangles and senile plaques are the two most characteristic lesions in the brains of Alzheimer's disease. The concentration of these lesions is correlated both with the severity of dementia and with deficiencies in the cholinergic system. The lesions can occur in the hippocampus of most aged individuals without dementia and their frequency appears to increase with age. Several studies have correlated the severity of Alzheimer's disease with the frequency of neuritic or senile plaques and with the reduction in presynaptic cholinergic markers in the cortex. The presynaptic marker refers to the axon terminals of a neuron which contain the vesicles filled with neurotransmitters, in this case acetylcholine. The results of such studies strongly suggest that loss of cholinergic neurons in the cortex plays an important role in the evolution of senile plaques.

In Alzheimer's disease, neurofibrillary tangles are present in large numbers. They occur in the cell body, where they displace other organelles, and appear as tangled masses of fiber-like elements sometimes irregularly dispersed throughout the cell and extending into the dendrites and axon of the neuron. The presence of these tangles in large numbers is not unique to Alzheimer's disease. They are also found in adult Down's syndrome, Guam Parkinsonian dementia complex (as well as neurologically intact younger individuals living in Guam), amyotrophic lateral sclerosis (ALS), and other neurological disorders. Despite great interest in the mechanism by which tangles are formed in both Alzheimer's disease and normal aging brains, attempts to characterize their biochemical composition have not yet succeeded. The neurofibers that make up the tangles consist of a pair of filaments wound around each other to form a paired helical filament (PHF).

The difficulty in studying the molecular nature of neurofibrillary tangles is evidenced by the recent discovery that these filaments or PHFs are highly insoluble in all of the reagents commonly used to solubilize proteins. A study by Selkoe of Harvard University suggests that the PHFs in Alzheimer's disease lesions contain a highly rigid large polymer whose molecules are held together by an extremely strong bond which makes it very insoluble. The evidence strongly suggests that covalent bonds cross-link specific amino acids of individual filaments into rigid polymer. Because of this unique insolubility characteristic of PHF, it would have been impossible to characterize the PHF proteins using the traditional methods of protein analysis.

The results of this work suggest that the cross-linking reaction takes place under the influence of a calcium dependent enzyme known as transglutaminase and that this type of reaction may play a general role in restructuring of cells during aging. Therefore, an increase in calcium concentration inside the neuron activates the enzyme transglutaminase, and this leads to cross-linking of proteins into insoluble large polymers such as PHF.

These findings are important and exciting because we now can begin to investigate the exact nature of the cross-linking bond and what causes it to form. The accumulation of these highly rigid and insoluble polymers may physically interfere with the normal axonal transport system and have serious consequences for the viability of the neuron. Although very little is known about the crucial events that initiate the formation of neurofibrillary tangles, it is now possible to manipulate the structural proteins of PHF and the proliferation of neural filaments in model systems.

Because PHF is one of the principal markers for Alzheimer's disease, it becomes an important scientific challenge to determine the crucial pathogenic event(s) underlying the cellular changes that lead to PHF formation. A group of Canadian scientists, Crapper-McLachlan and his colleagues, working on the assumption that the crucial pathogenic event involves either endogenous gene failure or an unidentified infectious agent, have proposed several hypotheses concerning the cellular changes underlying Alzheimer's disease. One of these suggests that changes in the blood-brain barrier may render neurons vulnerable to potentially toxic environmental agents such as silicon and aluminum. Circumstantial evidence supports this hypothesis. There is evidence of an age-related increase in brain aluminum concentration in humans. There is also considerable evidence that aluminum concentrations are elevated in the aged brain in association with neurons containing neurofibrillary tangles and with such degenerative diseases as Alzheimer's disease, Guam and Kii peninsula ALS, and Parkinsonian dementia complex.

Since there appears to be a high correlation between the presence of both PHF and aluminum, it is important to measure the precise distribution and concentration of aluminum within the cell. Scanning electron microscopy in conjunction with X-ray spectrometry provides an extremely sensitive analytical tool for identifying and localizing chemical elements with a neuron. Using this technique, Perl of the University of Vermont Medical School has shown that aluminum concentrates in the tangle-bearing neurons from the hippocampus of Alzheimer patients. Adjacent normal-appearing neurons from both Alzheimer patients and controls are free of any detectable aluminum.

In a more recent study, Perl, Gadjusek, and co-workers have analyzed tangle-bearing and tangle-free neurons from the hippocampus of Guamanian Chamorros with ALS and Parkinsonian dementia (PD) and neurologically normal controls. Their results show that aluminum accumulates within the tangle-bearing neurons of the hippocampus in the ALS and PD cases. The tangle-bearing neurons from the controls also had a high concentration of aluminum. Interestingly, the calcium-related X-ray emissions were higher in the tangle-bearing than in the tangle-free regions.

The constellation of ALS, PD, and extensive premature tangle formation among the Chamorros of Guam and the Japanese living in the Kii peninsula suggests that a common etiological factor or factors are involved in these conditions. In a careful study of the ecology, culture, and diet of the ALS and PD affected people, Gajdusek and his colleagues found these people to be indistinguishable from their unaffected neighbors except for the mineral content of their drinking water, which is extremely low in calcium and magnesium but very high in aluminum, silicon, titanium, chromium, iron, and manganese. However, the functional relationship between the mineral content of the drinking water and the appearance of tangle-bearing neurons or the pathogenesis of ALS, PD, or Alzheimer's disease is not understood. Clearly this suggests an important linkage that needs further study.

These results are extremely important because they provide a crucial beginning for piecing together the Alzheimer's disease puzzle. It is now possible to formulate testable hypotheses that can provide a common framework for interrelating a number of the discrete results discussed here. As has been indicated, an abnormality in the blood-brain barrier could allow neurotoxins such as aluminum to enter the brain and eventually the nerve cell. The aluminum as $Al(OH)^{2+}$, could then compete with the calcium-binding sites on specialized proteins, disrupting the normal homeostatic mechanism for maintaining low calcium levels within the cell. Increasing the net Ca^{2+} concentration in the cell could have a number of adverse consequences because of the key role Ca^{2+} plays as a second messenger within the cell. One of the most important of these effects is in axonal transport. Several studies have shown that Ca^{2+} is the critical factor for maintaining axonal transport. It has been suggested that some of the effects of metabolic inhibitors might be explained by the release of Ca^{2+}; the resulting high concentrations of free Ca^{2+} might inhibit axonal transport.

Changes in the concentrations of free Ca^{2+} inside the neuron can also influence other age-related changes.

Since the regulations of free calcium ions within the cell appears to be crucial for a number of cellular functions, understanding the biological mechanism of regulating calcium transport across membranes may provide a common framework for relating a number of these age-related changes.

In view of the available evidence, it is reasonable to propose than any change that seriously disturbs the delicately balanced homeostatic system of transporting Ca^{2+} would have serious consequences for the cell's ability to communicate, or even for its viability. Such a disruption of the system could be a consequence of exposure to a neurotoxin, pathogenic agent, aging, or other insults. Examination of the age-related change in the brain at this level of analysis would allow us to relate changes in the brain to changes in other systems, such as the cardiovascular, immune, and endocrine systems, and develop a better understanding of how cells communicate with each other in all systems. The approach would allow us to develop integrated theories of aging and better understand the biological mechanism of diseases such as Alzheimer's disease.

Conclusions

Although Alzheimer's disease was described nearly three-quarters of a century ago, its systematic study by the scientific community began only a few years ago.

During this brief span, our knowledge of this disease advanced from nearly total ignorance to a level where it is possible to begin formulating viable hypotheses, try possible treatment, and explain its causes.

The scientific community now has the tools, the knowledge base, and the means to solve the problem of Alzheimer's disease within the next decade. This remarkable progress in dementia research has been made with relatively small financial resources, because the availability of scientific knowledge, technology, and methods--the necessary bricks and mortar to start building a structure--has been more than equalled by increased interest and enthusiasm in the medical community and among the general public.

The U.S. Public Health Service, of which the National Institute on Aging is a part, has supported a significant proportion of basic biomedical research in this country during the last 30 years. A tremendous amount of this work has had direct and indirect bearing on our understanding of the basic mechanism of cells, including neurons. Using knowledge gained from such areas of science as biochemistry, cellular and molecular biology, bioengineering, physics, genetics, recombinant DNA, and cloning technology, it is now feasible to make rapid progress in understanding the cause of Alzheimer's disease as well as other disabling disorders of the aged.

Such past improvements in U.S. health care as reductions in childhood mortality have been based on public health prevention and measures such as control of infection, improved nutrition, and the lowered incidence of killer diseases such as cancer and cardiovascular disease. Current conditions, however, call for more mechanistic approaches based on understanding the structure and function of the body and of the disease processes that affect it. It is no longer sufficient to strive for the control of diseased organs; we must aim at understanding the disease mechanism.

It is important to emphasize that the linkage between basic research and health problems is probably closer in the neurosciences than in almost any other field of biomedical science. The need to establish the fundamental organizing principles and rules of operation of the human brain--especially in the elaborate systems governing coordination, performance, thought, memory, language, speech, hearing, and other quintessentially human faculties--logically precedes and forms the rational basis for working out solutions to the diseases and disorders that disrupt brain functions.

At the same time clinical observations provide a constant source of inspiration and information in this process. The first major advances in understanding the nature and origin of brain waves, in delineating the functions of deep temporal lobe structures, and in designing equipment to probe those structures arose from the desire to understand and treat epilepsy. Today, the observations that an aphasic stroke patient sometimes manages miraculous recovery of speech and language functions despite massive lesions, and that a dysarthric Huntington's disease patient in the late stages of dementia may suddenly produce a clear, well-spoken thought, offer clues to the still-hidden capabilities of the human brain and add impetus to the goal of fundamental understanding.

Mr. GORE. Thank you very much. We appreciate that.

The technical aspects of this research are difficult and complex, but we know that a solution will come from the work that you and others are doing, including the work being done here at this institution. And we are going to examine that in much greater detail in the other two hearings as well.

We're going to hold questions and now hear from Dr. Robert Cook-Deegan with the Office of Technology Assessment. And I might say to our guests here that the Office of Technology Assessment is a part of the legislative branch of Government, it is a part of the Congress, and it is the body that has responsibility for advising the Congress on technical and technological issues. They have done an outstanding job over the years. We now find people from other countries around the world coming to look at OTA—as we call it—as a model for their own efforts to set up bodies to advise them on how to handle this technological revolution that we are in.

I also want to say that I have formally requested, with indications from the Office of Technology Assessment that this request is going to receive priority attention, along with Congressman Larry Winn, the ranking Republican member of this full committee, and Congressman Don Fuqua and Congressman Pepper, a comprehensive analysis of the health care and research issues posed by Alzheimer's disease. This long-term study will examine in detail many of the problems that we are discussing here today.

And, Dr. Robert Cook-Deegan, you are one of the leading experts on this subject. We're delighted that you could come here to East Tennessee State today to be with us. If you could hold your formal statement relatively short, in view of the time constraints we have, we'll put your full statement in the record, and then we'll go to some questions. So, please proceed with your statement.

Dr. COOK-DEEGAN. Thank you, Mr. Chairman. And thank you for the opportunity to testify before the committee. My name is Dr. Robert Cook-Deegan. I am here primarily because I began research on Alzheimer's disease in 1976, at the University of Colorado, when I was a freshman medical student.

I would like to express my sympathy with those who spoke earlier this morning. I lost a grandmother to the disease about 6 weeks ago, as well.

I'm a physician working as a senior analyst at the Office of Technology Assessment. My primary work for the last 2 years has been for an assessment on the role of "Technology and Aging in America," that will be released in September or early October. As part of the OTA report, we identified five major disorders highly prevalent among older Americans that may become serious health problems for the Nation as the population ages, particularly as the oldest segments of the population expand more rapidly than other age groups.

The first and most threatening of the disorders we investigated was the group of diseases that cause irreversible loss of intellectual functions, or dementia. The most common cause of dementia is Alzheimer's disease, which accounts for an estimated 55 to 70 percent of cases and is combined with other causes in another 10 to 20 percent.

The prevalence of Alzheimer's disease is projected to rise rapidly over the next decade, as the number of individuals aged over 80 increases. This rise in prevalence portends an enormous and pervasive social burden, as family and Government agencies struggle to deal with the emotional, medical, and long-term care needs of millions of Americans.

Other witnesses will undoubtedly highlight the disastrous consequences of falling victim to Alzheimer's disease. I will concentrate my remarks on what the Federal Government might do to combat the anticipated onslaught of dementing illnesses.

When faced with a major health problem, Government can follow four basic strategies:

First, they can support acute medical care, including diagnosis and treatment of the condition.

Second, they can support long-term care, including medical and nonmedical services.

Third, they can encourage citizens to adopt behaviors that promote health and prevent developing the disorder or disorders.

Fourth, they can support research relevant to the condition.

In the case of Alzheimer's disease, the cause is not definitely known, there is no effective treatment, and there are no means of preventing it. Diagnostic practices have improved dramatically over the past decade as the disorder has received more attention from the medical community. While a few partially effective palliative treatments have been developed, no intervention can fully arrest the progression of the disease or eliminate its most severe symptoms. The campaign against Alzheimer's disease thus cannot rely on prevention or medical treatment until and unless scientific advances make them possible.

Government, for the time being, is effectively left with only two options, support of long-term care for those patients who develop the disorder and research intended to discover the means of prevention, new medical treatments, and optimal methods of delivering care.

Before moving on to remarks about research, I will merely note that Government activity in long-term care is presently a patchwork of uncoordinated services shared among local, State, and Federal agencies. And one of the groups least well served by the current situation is the cohort of patients afflicted by diseases that cause dementia and the families who care for them.

Current State and Federal policies on Medicaid, for example, include several disincentives to accepting confused patients into nursing homes and other long-term care services. A description of these biases is included in the long-term care chapter of the OTA assessment, and is too complex to elaborate here, except to note that the services provided are largely determined by what State and Federal agencies choose to pay for: in long-term care, as in other health care, form follows funding.

My final remark on long-term care is that the Veterans' Administration, because of the demographic characteristics of the veteran population, will receive the brunt of its share of patients with Alzheimer disease over the next decade, well in advance of the peak in the general population. This will stress the VA health care system, but it will also provide an opportunity for the Federal Government to investigate improved ways to care for patients with dementia. The VA is thus in an ideal position to lead the nation in developing comprehensive programs for dealing with dementia.

The areas of research that are supported by Government can be roughly divided into basic biological and medical research, clinical and applied research, and health services research. Solving the problems posed by dementing disorders will require a concerted and coordinated program that involves all three areas. By analogy to the health care sector, Federal support may well determine the outcome of any program of research: in biomedical research findings follow funding.

In the area of biomedical research, the Federal Government is the largest single supporter in the United States, funding 29 percent of it. The National Institutes of Health is the largest health agency in the United States and is known throughout the world as the keystone of the Nation's biomedical research infrastructure. The National Institute on Aging has been the lead agency in funding research on Alzheimer's disease for the past half decade, although the National Institute of Mental Health and the National Institute of Neurological and Communicative Diseases and Stroke also have significant programs on Alzheimer's disease and related disorders.

Other witnesses will describe the activities of NIA in greater detail—that is, the Aging Institute. However, I would like to point out that the NIA is in an excellent position to lead the effort to combat Alzheimer's disease, because its mandate—its legislative mandate—unlike other NIH institutes, includes provisions for supporting social and health services research in addition to basic biomedical research.

Successful eradication or treatment of Alzheimer's disease will require investigations in many diverse areas, including: epidemiology, pharmacology, basic neuroscience, and genetics, in addition to neurochemistry, immunology, and microbiology. The "ologies" could go on and on. The conquest of dementing illnesses may well serve as the rallying point for an important and rapidly expanding area in basic biology, namely neuroscience, just as cancer research fostered the dramatic expansion of molecular biology research in the 1970's. Support of basic neuroscience might prove to be one of the most important beneficial side products of any assault on neurological and psychiatric illnesses.

In the area of clinical and applied research, fruitful applications of basic biology relevant to Alzheimer's disease and related disorders will depend on continuing clinical and applied research. New treatments and methods of prevention will undoubtedly require continued study of the disease process, its consequences, and ways to alter the natural course of the diseases. Research in clinical neurology, psychiatry, clinical pharmacology, pathology, and, perhaps, neurosurgery, will all be needed. New techniques for refining the diagnosis of the various disorders that cause dementia are essential to progress in overcoming the ravages of dementing disorders.

In the area of health services research—health services research, the study of how to care for patients and how to finance such care, is a neglected area in general and is especially lacking with regard to the care of patients with dementia. In health services research it is less important to know the precise nature of the disease, be it Alzheimer's disease, stroke, or some other cause that causes the dementia, than to identify ways to take care of patients whose intellectual and emotional capacities have been eroded by disease.

Even rudimentary statistics on how many people are affected, where they are cared for, and the type and stage of illness are extremely uncertain. The wide range of figures noted on the OTA fact sheet that is included with my written testimony reflects this uncertainty. The study of various techniques of caring for patients with dementia in acute and long-term care settings is in its infancy. In our judgment a Federal commitment to support of health services research on home care, respite care, family care, day care, foster care, board and care facilities, and domiciliary facilities, as well as nursing homes and hospitals, will be important in dealing with problems posed by dementia for at least the next decade. There is no alternative to long-term care of patients with dementia in the absence of medical breakthroughs.

Health services research must include alternative mechanisms for financing care as well as comparative testing of various methods for taking care of patients. The mechanisms for funding care range from complete public subsidy to subsidy based on the need to complete private financing. Alternatives for private payment include long-term care insurance, life-care communities, individual retirement accounts, equity conversion of house to cash, and incentives for voluntarism and family care.

The multiplicity of options noted, both in settings of care and methods of paying for them, suggest the complexity that must be addressed in identifying optimum alternatives for taking care of patients with irreversible dementia.

In conclusion, the problems caused by disorders that cause dementia, including Alzheimer's disease, are large, expensive, and rapidly growing. Federal policy on research is essential for addressing those problems, in hopes of either finding treatments, means of prevention, or optimal methods of caring for those who fall victim to dementing illnesses. A balanced attack will include support of basic biomedical research, clinical and applied research, and health services research.

Thank you.

[Applause.]

[The prepared statement of Dr. Cook-Deegan follows:]

TESTIMONY OF ROBERT M. COOK-DEEGAN, M.D.
SENIOR ANALYST, BIOLOGICAL APPLICATIONS PROGRAM
OFFICE OF TECHNOLOGY ASSESSMENT, U. S. CONGRESS
BEFORE THE SUBCOMMITTEE ON INVESTIGATIONS AND OVERSIGHT
OF THE COMMITTEE ON SCIENCE AND TECHNOLOGY
U. S. HOUSE OF REPRESENTATIVES

ALZHEIMER DISEASE AND OTHER DISORDERS CAUSING DEMENTIA

August 30, 1984

East Tennessee State University
Johnson City, Tennessee

Mr. Chairman,

Thank you for the opportunity to testify before the subcommittee. My name is Dr. Robert Cook-Deegan. I am a physician working as a Senior Analyst at the Congressional Office of Technology Assessment. My primary work for the past two years has been for an assessment on the role of ***Technology and Aging in America*** that will be released in September or early October. As part of the OTA report, we identified five major disorders highly prevalent among older Americans that may become serious health problems for the nation as the population ages, particularly as the oldest segments of the population expand more rapidly than other age groups. The first and most threatening of the disorders we investigated was the group of diseases that cause irreversible loss of intellectual functions, or **dementia.** The most common cause of dementia is Alzheimer disease, which accounts for an estimated 50 to 70 percent of cases, and is combined with other causes in another 10 to 20 percent.

The prevalence of Alzheimer disease is projected to rise rapidly over the next decade as the number of individuals over age 80 increases. This rise in prevalence portends an enormous and pervasive social burden as families and government agencies struggle to deal with the emotional, medical, and long-term care needs of millions of Americans.

Other witnesses will undoubtedly highlight the disastrous consequences of falling victim to Alzheimer disease. I will concentrate my remarks on what the Federal government might take to combat the anticipated onslaught of dementing illnesses.

When faced with a major health problem, governments can follow four basic strategies:

1 They can support acute medical care, including diagnosis and treatment of the condition.

2 They can support long-term care, including medical and non-medical services.

3 They can encourage citizens to adopt behaviors that promote health and prevent developing the disorder or disorders.

4 They can support research relevant to the condition.

In the case of Alzheimer disease, the cause is not definitely known, there is no effective treatment, and there are no means of preventing it. Diagnostic practices have improved dramatically over the past decade as the disorder has received more attention from the medical community. While a few partially effective palliative treatments have been developed, no intervention can fully arrest the progression of the disease or eliminate its most severe symptoms. The campaign against Alzheimer disease thus cannot rely on prevention or medical treatment until and unless scientific advances make them possible.

Government, for the time being, is effectively left with only two options, support of long-term care for those patients who develop the disorder, and research intended to discover means of prevention, new medical treatments, and optimal methods of delivering care.

LONG-TERM CARE

Before moving on to remarks about research, I will merely note that government activity in long-term care is presently a patchwork of uncoordinated services shared among local, State, and Federal agencies, and one of the groups least well served by the current situation is the cohort of patients afflicted by diseases that cause dementia. Current State and Federal policies on Medicaid, for example, include several disincentives to accepting confused patients into nursing homes and other long-term care services. A description of these biases is included in the Long-Term Care chapter of the OTA assessment, and is too complex to elaborate here, except to note that the services provided are largely determined by what State and Federal agencies choose to pay for: in long-term care, as in other health care, "form follows funding."

My final remark on long-term care is that the Veterans Administration, because of the demographic characteristics of the veteran population, will receive the brunt of its share of patients with Alzheimer disease over the next decade, well in advance of the peak in the general population. This will stress the VA health care system, but it will also provide an opportunity for the Federal Government to investigate improved ways to care for patients with dementia. The VA is thus in an ideal position to lead the Nation in developing comprehensive programs for dealing with dementia.

RESEARCH

The areas of research that are supported by government can be roughly divided into basic biological and medical research, clinical and applied research, and health services research. Solving the problems posed by dementing disorders will require a concerted and coordinated program that involves all three areas. By analogy to the health care sector, Federal support may well determine the outcome of any program of research: in biomedical research "findings follow funding."

Basic Biomedical Research

The Federal Government is the largest single supporter of health-related research in the United States, funding 29 percent of it. The National Institutes of Health is the largest health research agency in the United States, and is known throughout the world as the keystone of the Nation's biomedical research infrastructure. The National Institute on Aging (NIA) has been the lead agency in funding research on Alzheimer disease for the past half decade, although the National Institute of Mental Health and the National Institute of Neurological and Communicative Disorders and Stroke also have significant research programs on Alzheimer disease and related disorders.

Other witnesses will describe the activities at NIA in greater detail.* However, I would like to point out that NIA is in an excellent position to lead the effort to combat Alzheimer disease because its mandate, unlike other NIH institutes, includes provisions for supporting social and health services research, in addition to basic biomedical research.

Successful eradication or treatment of Alzheimer disease will require investigations in many diverse areas, including epidemiology, pharmacology, basic neuroscience, and genetics, in addition to neurochemistry, immunology, and microbiology. The conquest of dementing illnesses may well serve as the rallying point for an important and rapidly expanding area in basic biology, neuroscience, just as cancer research fostered the dramatic expansion of molecular biological research in the 1970's.

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* OTA was assisted in its assessment ***Technology and Aging in America*** by Dr. Zaven Khachaturian of NIA.

Support of basic neuroscience might prove to be one of the most important beneficial side products of any assault on neurological and psychiatric illnesses.

Clinical and Applied Research

Fruitful applications of basic biology relevant to Alzheimer disease and related disorders will depend on continuing clinical and applied research. New treatments and methods of prevention will undoubtedly require continued study of the diseases process, its consequences, and ways to alter the natural course of the diseases. Research in clinical neurology, psychiatry, clinical pharmacology, pathology and, perhaps, neurosurgery will be needed. New techniques for refining the diagnosis of the various disorders that cause dementia are essential to progress in overcoming the ravages of dementing disorders.

Health Services Research

Health services research, the study of how to care for patients and how to finance such care, is a neglected area in general, and is especially lacking with regard to the care of patients with dementia. In health services research, it is less important to know the precise nature of the disease (be it Alzheimer disease or stroke) that casues the dementia than to identify ways to take care of patients whose intellectual and emotional capacities have been eroded by disease (whatever the origin).

Even rudimentary statistics on how many people are affected, where they are cared for, and the type and stage of illness are extremely uncertain. (The wide range of figures noted on the OTA Fact Sheet included with my written testimony reflects this uncertainty.) The study of various techniques of caring for patients with dementia in acute and long-term care settings is in its infancy. In our judgment, a Federal commitment to support of health services research on home care, respite care, family care, day care, foster care, board and care facilities, and domiciliary facilities, as well as nursing homes and hospitals will be important in dealing with the problems posed by dementia for at least the next decade. There is no alternative to long-term care of patients with dementia in the absence of medical breakthroughs.

Health services research must include alternative mechanisms for financing care as well as comparative testing of various methods for taking care of patients. The mechanisms for funding care range from complete public subsidy to subsidy based on need to complete private financing. Alternatives for private payment include long-term care insurance, life-care communities, individual retirement accounts, equity conversion plans, and incentives for voluntarism and family care.

The multiplicity of options noted, both in settings of care and methods of paying for it suggest the complexity that must be addressed in identifying optimum alternatives for taking care of patients with irreversible dementia.

CONCLUSION

The problems caused by disorders that cause dementia, including Alzheimer disease, are large, expensive, and rapidly growing. Federal policy on research is essential for addressing those problems, in hopes of either finding treatments, means of prevention, or optimal methods of caring for those who fall victim to dementing illnesses. A balanced attack will include support of basic biomedical research, clinical and applied research, and health services research.

WRITTEN SUPPLEMENT

for

TESTIMONY OF ROBERT M. COOK-DEEGAN, M.D.
SENIOR ANALYST, BIOLOGICAL APPLICATIONS PROGRAM
OFFICE OF TECHNOLOGY ASSESSMENT, U. S. CONGRESS
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ALZHEIMER DISEASE AND OTHER DISORDERS CAUSING DEMENTIA

August 30, 1984

East Tennessee State University
Johnson City, Tennessee

OTA FACT SHEET ON ALZHEIMER DISEASE

How big is the problem?

- Alzheimer disease affects 5 to 15 percent of those over 65 (1.2 to 4 million Americans).
- Alzheimer disease is more common with advancing age, affecting more than 20 percent of those over age 80.
- Alzheimer disease imposes severe financial, medical, and and emotional stresses on families.
  - A full diagnostic evaluation costs $300 to $2000.
  - Families bear fractions of diagnostic and therapeutic costs, and almost half of the overall cost of long-term care directly out-of-pocket.
  - Families suffer loss of a productive member, often leading to sudden and substantial reduction in income.
- Alzheimer Disease accounts for an estimated 30 to 50 percent of those in nursing homes.
- Alzheimer disease is the most frequent cause of institutionalization for long-term care.
- Federal funds for long-term care in 1980 were $12 billion ($10.4 from Medicaid, $0.4 from Medicare, and $1.1 from the VA). These costs, under present arrangements, will escalate to $24 billion by 1985, and $43 billion by 1990. Alzheimer disease accounts for 30 to 50 percent of this spending.
- Alzheimer disease accounts for an estimated 50 to 75 percent of dementia ("senility"). The remaining fraction is due to cardiovascular disorders, stroke-related diseases, and more than a dozen treatable causes of dementia.
- Alzheimer disease is often misdiagnosed; 8 to 23 percent of those said to have this disease may actually have depression or other treatable disorders.
- Chances are better than 50 percent that one of one's grandparents would develop the disease if all lived past age 65.

What is it?

Alzheimer disease is an organic brain disease named after the German physician, Alois Alzheimer, who first described it in 1906. Alzheimer disease causes progressive loss of mental functions over a period of years. Its clinical progression is usually divided into three stages:

1. Mild cognitive deterioration is usually first detected by loss of memory for recent events. Difficulties in speech and word-finding; loss of the ability to "think through" complex actions or interpret complex stimuli soon follow. An example of a mental ability that is often lost is the ability to do arithmetic.
2. This is followed by worsening cognitive deterioration and personality changes, often including irritability and severe disorientation as to time and place.
3. Finally, the patient shows complete loss of intellectual abilities, leading to a state of vegetation. This includes the inability to speak or recognize even close relatives, and complete loss of "world knowledge."

It is highly probable that what is now called "Alzheimer disease" or "senile dementia of the Alzheimer type" is a group of several distinct diseases, with different causes and mechanisms. There is tremendous variation in the duration of the illness and its rate of progression. Younger patients tend to have more severe cases which progress more rapidly. Alzheimer disease can last from a few years to a few decades.

How is it diagnosed?

The diagnosis of Alzheimer disease can now be made only by excluding other causes of dementia. Diagnosis is certain only when made at autopsy, by finding a profusion of the three pathologic indicators of Alzheimer disease:

- "Senile" or Alzheimer plaques (neuritic plaques) -- accumulations of proteins and cellular debris also found in other diseases, but not in such numbers or in the variety of anatomic locations as in Alzheimer disease.
- Neurofibrillary tangles -- accumulations of fiber-like proteins inside of cells, also found in several other disease states, but with a different distribution and in lesser numbers.
- Granulovacuolar bodies -- enclosed bundles (vacuoles) of material of uncertain origin found inside cells. They are restricted to specific regions of the brain, and are rare in other diseases.

Prepared by the Office of Technology Assessment (OTA), U. S. Congress,
as part of its assessment of Technology and Aging in America

OTA FACT SHEET ON ALZHEIMER DISEASE

None of the pathologic changes is unique to Alzheimer disease. The pathologic diagnosis is based on seeing a large number of the plaques, tangles, and granulovacuolar bodies in places not usually associated with other diseases. Correlation with clinical symptoms is essential to definitive diagnosis.

What are other important points?

- It is not clear whether Alzheimer disease is a consequence of aging or a specific disease which affects only a susceptible population. Present medical belief favors the latter hypothesis; historically, the relative dominance of these beliefs has alternated.
- All statistics on incidence and prevalence are highly uncertain because:
  - Alzheimer disease patients die with the disease, not of it. This leads to under-reporting on death certificates.
  - Patterns of diagnosis of psychiatric and neurological diseases have changed drastically in the last decade. "Alzheimer disease" used to refer only to those patients affected before the arbitrary age of 65.
  - Alzheimer disease patients often are not referred to a neurologist or psychiatrist for specific diagnosis. Terms such as "senility," "cerebral arteriosclerosis," "organic brain syndrome," "chronic brain syndrome," and "dementia" often serve as substitutes for an accurate and specific diagnosis. Nonspecific and uncertain diagnosis make data on dementia unreliable, especially when not gathered by neurology or psychiatry departments.
- There is no effective treatment at present.
- There is no specific "marker" for the disease. Thus the diagnosis is made by excluding other causes of poor mental function. This leads to diagnostic error. There is a need for accurate and cost-effective diagnosis.
- The disease starts as the patient's and ends as the family's: the patient's medical problem soon becomes a financial and emotional drain on the family. Many of the most distressing effects of the disease are borne by friends and relatives, not by the patient.
- Self-help groups, such as the Alzheimer disease and Related Disorders Association, are trying to aid in family support and as information clearinghouses.
- There are no effective means of prevention.
- Because the ability of the patient to decide for himself or herself is often questionable, the procedure for obtaining informed consent may be unclear, leading to legal and ethical problems in research and treatment.

What are some possible causes?

- Loss of nerve cells in specific areas of the brain (nucleus basalis of Meynert, locus ceruleus) may be one cause, analogous to Parkinson disease. The reason for the loss is unknown.
- Genetic factors may account for 10 to 60 percent of cases. The wide range in estimates is due to scientific uncertainty. There is definitely at least one genetic form, in which an affected person's sons or daughters are each at 50 percent risk, but the prevalence of the genetic form is controversial.
- An atypical infectious agent ("slow virus"), analogous to that which causes some other dementing diseases, has been suggested. The other diseases are caused by unusual infectious agents that remain dormant for years, and then cause unremitting neurological illnesses which terminate in death after a clinical course of one to a few years. Alzheimer disease is similar to these other diseases in several respects, but there is only circumstantial evidence of causal similarity at present.
- Condensation of fibrillar proteins in neurons, of unknown cause, may lead to interference with normal nerve-cell function, eventually resulting in cell death.
- Aluminum is found in high concentrations in the plaques of those affected with Alzheimer Disease. One hypothesis is that cell death is due to some variety of "aluminum poisoning." There is no agreement on whether the aluminum accumulation is a cause or an effect of cell death and biochemical changes in Alzheimer plaques.
- It has also been suggested that Alzheimer disease may be an immune disorder, caused by the immune system reacting against the brain, leading to cell death. This would be analogous to other diseases such as systemic lupus erythematosus or myasthenia gravis.
- A biochemical aberration of calcium metabolism may lead to insufficient release of neurotransmitter.

OTA FACT SHEET ON ALZHEIMER DISEASE

- o There may be a disorder of glycolytic biochemical pathways (pyruvate dehydrogenase) leading to decreased calcium influx into the cell and insufficient release of neurotransmitter.
- o Disorders of neurotransmitters other than acetylcholine may be involved as well. Those suggested include:
  - somatostatin,
  - norepinephrine (locus ceruleus),
  - vasopressin, and
  - endogenous opioid peptides. This last is supported by clinical reports of improvement by treatment with naloxone, a chemical blocker of opiate receptors.

What potential therapies are under investigation?

- o Stimulate acetylcholine synthesis (lecithin, choline) -- not very successful to date.
- o Administer piracetam, a drug which improves some aspects of mental function, alone or in combination with other drugs.
- o Use drugs that mimic the effect of acetylcholine, either alone or in combination.
- o Stimulate acetylcholine release with 4 amino-pyridine and related agents
- o Increase the duration of action of acetylcholine by inhibiting breakdown by anticholinesterase drug therapy. The best example is physostigmine.
- o Chelate aluminum to prevent toxicity with fluoride, tetracycline, or deferoxamine.
- o Use naloxone to block opiate receptors.
- o Use vasopressin to improve memory.
- o Breathe hyperbaric oxygen (not very successful).
- o Use ergoloid mesylates (Hydergine -- only marginally effective, used widely in Europe).

Of these treatments, those affecting acetylcholine have shown moderate, but inconsistent improvement. Ergoloid mesylates administration has been shown to have an effect in numerous trials, but the effect is so minor that it may not be clinically relevant.

What are some potential developments in the long run?

- o Research may yield a specific diagnostic test for Alzheimer disease. Such a test could develop from biochemical tests on tissue from brain biopsies, chemical metabolites in cerebrospinal fluid, or monoclonal antibodies to proteins that are possibly specific to Alzheimer disease.
- o Use of nerve-cell implants (so-called brain-cell transplants) is promising, but many technical obstacles remain. The treatment has worked in animals with a disease resembling Parkinson disease, and human grafts have grown successfully in experiments conducted in Scandinavia. The human experiments have not yet been clinically useful, and many problems regarding what tissue to use as a source of implantable nerve cells must be solved. Successful implant therapy for Alzheimer disease also awaits knowledge of the nerve cells that need to be replaced.
- o Understanding of the biochemical and neurotransmitter defects may allow specifically targeted therapy, based on the physiology of the affected parts of the brain. One promising avenue to learning about this is through development of animal models based on finding chemicals that specifically destroy certain parts of the brain or particular types of nerve cells.
- o Understanding a cause or causes (whether viruses, other infectious agents, or environmental toxins) may permit effective prevention.
- o Identification of genetic markers, analogous to those known for sickle cell disease or Huntington disease, would prove useful for investigating the putative molecular defect, as well as helping in diagnosis of members of affected families for family and personal planning.
- o Genetic treatments may be possible, based on studying human genes in other species (such as hamsters or mice), and identification and specific study of those genes, if any, that predispose to dementia. It may also be possible to deliberately engineer nerve cells with needed characteristics to replace or supplement cells that have died.

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DEMENTIA: SOCIAL PROBLEM, MEDICAL ENIGMA, AND FEDERAL BURDEN

Dementia is one of the most important disorders leading to chronic disability among older Americans. It affects fewer people than osteoarthritis, and is not a major direct cause of death like cancer or cardiovascular disease. In epidemiological studies, however, life expectancy is reduced by half among demented patients, making it the fourth or fifth highest cause of death among older people. Yet the effects of dementia are so devastating to the patient and his or her family, and its prevalence is increasing so rapidly, that medical writer Lewis Thomas has called it the "disease of the century."

Causes and Prevalence

Dementia is a serious social problem, a difficult medical enigma, and a major Federal expense. The loss of intellectual and other "higher" functions that is referred to as dementia can be caused by many different diseases and environmental insults.

[Table 1, causes of dementia]

The most common cause of dementia is Alzheimer disease, which accounts for 50 to 75 percent of all cases. Up to 20 percent of patients with dementia have a disease that is treatable, and proper treatment can improve mental function in many cases.* Another 15 to 25 percent have dementia due to cardiovascular disease, stroke, or an accumulation of "mini-strokes." Another common cause is inappropriate drug treatment for other illnesses; many drugs used to treat cardiovascular diseases, psychiatric diseases, and insomnia lead to poor mental function. Mental function often improves when the offending medication is withdrawn, and failure to distinguish drug-induced dementia from true irreversible dementia can result in unnecessary mental confusion and inappropriate care. A substantial, but unquantified, number of older people suffer from transient confusion that spontaneously resolves or goes away with medical treatment of another condition. Such acute confusional states pose a different set of problems in dealing with patients than does chronic dementia; acute dementia is not explored further in this discussion. Although there are many causes of irreversible dementia, the functioning of affected patients is generally similar regardless of cause; daily care techniques are thus also similar. Because stroke as a cause of dementia is, in general, treated as is stroke causing other symptoms, a scientific review of this type of dementia is not included here. In contrast, Alzheimer disease is the largest single cause of irreversible dementia, and little is known about its cause or means of prevention.

Alzheimer disease, first described in 1906 by German physician Alois Alzheimer, leads to progressive loss of brain functions over a period of years. The cause is unknown and there is no effective standard medical treatment.

Alzheimer disease affects between 1.2 and 4 million Americans; its true prevalence cannot be accurately determined because of variations in regional and historical methods of diagnosis of dementing illnesses. The term "Alzheimer disease," for example, was until recently restricted to those cases beginning before the arbitrary age of 65. Best estimates suggest that severe dementia affects approximately 2.5 percent of those over 65, and moderate and mild dementia probably afflicts another 2.5 to 10 percent. The disease becomes more common with age, affecting 2 to 30 percent of those over 80. More than half of all nursing home residents suffer from dementia.

Effects on Patients and Their Families

The patient is usually aware of intellectual decline in the early stages of the illness. As the disease progresses, the patient loses touch with the outside world, first through loss of memory and intellectual functions, then through loss of personality and speech, and finally through loss of the ability to recognize even close relatives and spouses.

The effect of dementia on family and friends is devastating. Families care for the vast majority of patients with dementia. For the most part, only those in the final stages of severe dementia are placed in nursing homes or other institutions of long-term care. Families and friends are estimated to attend to 80 percent of the long-term care needs of patients in the United States. [See Chapter 7 in the full assessment report ***Technology and Aging in America***, on Long-Term Care.]

Beyond the financial and medical burdens imposed by those who develop this disease are the severe emotional strains faced by families and friends. The progressive intellectual deterioration of a close loved one is a tragic experience that can only be fully understood by those who have gone through the process; it has been described by one family member as a "funeral that lasts for years." The arduous task of caring for patients with dementia is tellingly described in a book called The 36-Hour Day.

Clinical and Scientific Background

The clinical changes of Alzheimer disease are associated with physical and biochemical changes in the brain. Accumulations of abnormal proteins and other material are found in "plaques" in the cerebral cortex of the brain, as are accumulations of abnormal fiber-like proteins within nerve cells. Specific populations of nerve cells in the nucleus basalis of Meynert, near the base of the brain, die for unknown reasons. The death of these cells is postulated to be related to the clinical symptoms.

Patients die with this disease, not of it. Patients with dementia have only half the usual life expectancy for a given age; not because it kills in and of itself, but because patients become susceptible to other diseases as a consequence of being unable to take care of themselves and because of immobility caused by the end stages of the disease. At present, its cause is unknown, and there are no means of prevention. The absence of effective medical intervention has diminished the incentives to physicians to provide accurate and well-documented diagnosis; if the cause of illness does not affect treatment choices, there is less reason to identify it. Greater awareness of the fact that up to 20 percent of dementia is treatable could improve physician attention to accurate diagnosis of dementia. Growing public awareness of Alzheimer disease has drawn medical attention to the disorder, yet one of the most important reasons for accurate diagnosis is often overlooked -- families of patients know that something is wrong, but do not know what. The specific diagnosis of organic brain disease is unwelcome, but may reduce the anxiety and guilt felt by patients' families by identifying the problem as an organic brain disease rather than a problem of family or interpersonal dynamics.

The widespread belief in dementia as a normal concomitant of aging has historically served to lessen physician interest in the disease. The problem of diagnosis was further hampered by physicians who inaccurately ascribed dementia to atherosclerosis or other cardiovascular dysfunction. Diagnostic methods are now changing rapidly, and heightened awareness has led to a dramatic increase in specific diagnosis by physicians. Nevertheless, there is great uncertainty about the real prevalence of Alzheimer disease.

Although there is a genetic form of the disease, which runs in families (much like such genetic diseases as Huntington disease), it is not yet certain how many cases of Alzheimer disease are of the familial type; estimates of the number of patients who have a genetic form vary from fewer than 10 percent to more than 60 percent. Determining the genetic basis of Alzheimer disease is an important objective for research, especially if a marker for the disorder could be found to aid in diagnosis in affected families. Various causes of the disease have been postulated, including unusual infectious agents ("slow viruses"), aluminum toxicity, reaction of the body against itself (autoimmune disorder), and various biochemical abnormalities. Many research leads are promising, but none are yet conclusive.

Federal Roles Regarding Dementia

The basis of Federal concern about Alzheimer disease is not only in its prevention and treatment, but in payment for the care of patients. Because dementing illnesses account for admission of most nursing-home residents, they are a major aspect of long-term care and institutionalization. [See Chapter 7 in the full assessment report ***Technology and Aging in America*** on Long-Term Care.] The Health Care Financing Administration has estimated that the Federal Government spends over $6 billion annually in nursing home costs alone for those affected with Alzheimer disease (approximately 30 percent of the total Federal outlay -- 1982 data). An additional $2 billion to $4 billion in Federal funds are spent each year on acute hospital, home, and rehabilitative care for these patients.

Federal concern about Alzheimer disease is intensifying as its prevalence increases with the rapid growth of the oldest segments of the American population. The magnitude of health care costs, especially for long-term care, has focused new attention on the scientific and medical need to study this disease.

Concern among physicians, researchers, and other health care professionals has led to rapid advances in the study of Alzheimer disease in the last decade. A significant factor has been the creation of the National Institute on Aging (NIA), which has made a major commitment to the study of dementia. In 1984, for example, NIA earmarked $3.5 million to establish up to five "centers of excellence" for the study of Alzheimer disease in response to congressional appropriations. The National Institute of Neurological and Communicative Diseases and Stroke (NINCDS) and the National Institute of Mental Health (NIMH) have also increased research on dementia. Recent discoveries of specific nerve-cell loss, of the nature of the anatomic abnormalities, possible infectious agents, and of biochemical changes have all emerged from Federally funded research during the last decade.

Recent findings in neuroscience have led to experimental drug therapies directed at particular chemical nerve-cell connections in the brain. There has been preliminary success with drugs that allow the neurotransmitter acetylcholine to work longer at its anatomic site. The neuropeptide vasopressin has been shown to improve memory slightly in some patients with Alzheimer disease. The drug naloxone, which is used to treat some forms of acute opiate overdose, has also been reported effective in one small study, but following studies have not shown a significant effect. Tests have begun on drugs that might increase the efficiency of releasing acetylcholine into its active site by altering calcium metabolism and other biochemical pathways. Federal policy regarding support of randomized clinical trials of various therapies will affect the pace at which effective drug treatments are identified and accepted by the medical community. Preliminary findings offer hope for at least a partially effective symptomatic treatment. Solving the problem of dementia may well depend on developing effective means of prevention, which will, in turn, depend on understanding its cause, and the mechanisms of its progression.

There is also a role for the Government in protecting those who have Alzheimer disease. For example, who is to decide when it is proper to conduct research on a demented patient? The patient is by definition not able to give informed consent, which requires understanding of the purpose and process of experimentation. Obtaining informed consent can be a barrier to scientific investigation of just those treatments that are so needed. The dual role of the Federal Government in both preserving civil rights and supporting most biomedical research suggests that informed consent for Alzheimer Disease research may be an emerging Federal issue.

Treatment and prevention of this dread disease can only come from investment in neuroscience research. The United States has a social, financial, and medical stake in the solution of this major health problem. The Federal Government already sponsors research, pays for acute care and diagnosis through Medicare, and is responsible for long-term care through Medicaid. Cost savings in health care for the rising population of demented patients depend on finding new, effective, and inexpensive treatments for dementia and determining methods of prevention for Alzheimer disease and other dementing illnesses.

TABLE 1: COMMON REVERSIBLE CAUSES OF DEMENTIA

Drugs
- Sleeping pills and sedatives
- Other psychopharmaceuticals
- Anti-parkinson drugs
- Other neurological drugs
- Anti-hypertensive drugs
- Other cardiovascular drugs
- Opiate analgesics
- Many others

Depression and Psychosis

Infections
- Colds
- Flu
- Lung (pneumonia)
- Brain
- Brain membranes
- Blood
- Kidney
- Digestive tract
- Many others

Stroke (often at least partially reversible)
- Blood clots in or around the brain
- Occlusion of blood vessels feeding the brain

Heart Failure

Disorders of Endocrine Function
- Thyroid hormone
- Parathyroid hormone
- Hormones and substances affecting blood volume control
- Stress-related hormones
- Others

Nutritional Disorders
- Vitamin B_{12} deficiency
- Folate deficiency
- Protein deficiency

Anemia

Metabolic and Electrolytic Imbalances
- Calcium
- Sodium
- Potassium
- Others

Trauma
- Head injury
- Shock

Toxicity
- Occupational exposure
- Environmental exposure (lead, carbon monoxide)
- Household exposure (pesticides)

Mr. Gore. Thank you. Thank you both for your statements.

I'm going to call on Senator Pepper for any questions that he might have, but I want you to both know how appreciative we are of your work here.

And, Dr. Cook-Deegan, we take note of the personal experience your family has had with this. And I'm glad that you are scheduled to be the project officer for this comprehensive report by the Office of Technology Assessment.

Senator Pepper.

Mr. Pepper. Gentlemen, you've stirred our minds with the possibilities you've outlined for improving the situation in the future. As clear as you've made the analysis, it indicates that unless we can find some way to speed up the process, it's still going to be a good while before we're going to find the answer to this problem.

Have you any suggestions as to how Government, with its research facility, with the funds that it can make available, can expedite the discovery of the cause and cure of this terrible disease?

Either one. I've asked either one of you doctors.

And, by the way, both of you made a magnificent statement, and we're going to study particularly, in the future, what you've said. You've made a very thorough analysis of the whole technical problem presented, revealing, of course, the magnitude of the problem in so many areas which have to be explored before, perhaps, you'll find the answer to the problem.

But is there anything we can do to speed up the process?

Dr. Khachaturian. Congressman Pepper, as you know, since you have had a tremendous amount of experience, since you are, in many ways, responsible for the inception of the National Institute on Aging and are familiar with how NIH works, it is very difficult to predict how science is going to progress. But one thing is absolutely clear, is that the rate at which new discoveries are coming is increasing. For instance, years ago it used to take years between major discoveries; right now it's taking literally weeks between major discoveries.

Mr. Pepper. Are we putting enough funds into the research program?

Dr. Khachaturian. That's a difficult question to answer because it's not just the research funds, it's a question of having the right questions to ask, having the right technology, having the right amount of scientists to be interested in a given area. We are at the verge of creating that environment now with respect to aging and Alzheimer's disease. We're at the point of getting a critical mass of scientists interested in the problem. It's taken approximately 10 years to come to that point. Things are beginning to happen now.

Mr. Pepper. Well, would you say we now have a critical mass available?

Dr. Khachaturian. As I said we're getting there gradually.

Mr. Pepper. You know, we can find a way to spend an awful lot of money finding a good new weapon and all that sort of thing. And I just wonder, what do you think, Dr. Cook-Deegan: Are we really going after this problem in the wholehearted way in which we should, and can we do more?

Dr. Cook-Deegan. Well, I'll make a couple of observations. No. 1, I think that this is one of the major public health problems in the next half century. And frankly, no, I don't think there is enough funding going to this area. I don't think it's been made a national priority until the last 5 years. NIA took the lead in identifying Alzheimer's disease as a priority only about—well, the NIA was only created in 1974, and the programs really didn't gear up until the late 1970's, and they didn't surpass the NINCDS, and other NIH agencies, until about 5 years ago in funding for it.

I'll speak as an individual here rather than as a member of OTA. But when I first applied for a grant in 1976, as a medical student, Alzheimer's disease was something of a black sheep in the family; it was hard to get funding to do research on Alzheimer's disease. That has turned around quite a bit in the last, oh, 8, 9, years, especially in the last 5 years. But in my opinion this is a priority item and could use some more funding.

My only concern in funding it is to make sure that the money is spent productively as it's being appropriated.

Mr. **Pepper**. You know, sometimes I sort of have a feeling, sometimes when you people are being questioned by committees of the Congress, they think you're trying to steal money from the Treasury or something—you're there to raid the Treasury or something. I often wondered if we, maybe, might well approach you all by saying, "We want to know how much money can we wisely and effectively and efficiently spend trying to find the solution to this terrible problem."

Do you think you all are getting all the funds you can wisely, and honestly, and effectively spend in research?

Dr. **Cook-Deegan**. I think that, in the long run, if the current levels of funding were to go on for a long period of time, no. I think that it should be made more of a priority item. My concern is that the commitment should be for a long period of time and that—I'm thinking of cancer research, for example, that the war on cancer was declared in 1971, and the results of those funding decisions made in the early 1970's didn't really come to fruition until half a decade or a decade later. I think it should be a long-term commitment. I think that over the next decade, two decades, we have to get a start on it now. And I think that if a long-term commitment is made by Congress, by the executive branch, there is going to be room to spend a lot of money on this priority item. And I think the science will follow the funding, as I've said. And yes, I think we could use more.

Mr. **Pepper**. You're talking about the next one or two decades. How many people do you suppose will have Alzheimer's Disease in the next two decades?

Dr. **Cook-Deegan**. Too many.

Mr. **Pepper**. There are lots of them, aren't there?

Dr. **Cook-Deegan**. No, and I'm saying that I think there is room——

Mr. **Pepper**. One way you spend lives, and you spend the tragedy of families' experience, you spend what it costs to care for these people the best we can. The other way you spend the money in research that's helpful and productive, maybe, and saving in its character. So, I think you pose some serious decisions for both Congress and the country to make.

Dr. **Cook-Deegan**. I would make the observation that I think that the nation is spending at least $5 to $10 billion a year right now on Alzheimer's disease alone; and yet, the research funding for it is only in the range of—well, it's less than $50 million, certainly.

Mr. **Pepper**. I wonder, Doctor, if the amount of money we spend in trying to provide humane care, long-term care, for these victims may not be more than we're going to be spending in research——

Dr. **Cook-Deegan**. I think it will be.

Mr. **Pepper** [continuing]. To save that much.

Dr. **Cook-Deegan**. I think that it will be.

Mr. **Pepper**. Thank you very much.

Mr. **Gore**. That's an important point, if the cost of the long-term care is going to exceed the increment for finding a cure for this. And that's certainly an important point.

Doctor, you wanted to make a point?

Dr. **Khachaturian**. Yes. I want to make the point that the estimated cost for total health care for the elderly in 1983 was $120 billion. For nursing home care in 1983, it was $27 billion. It is estimated that within 10 years that amount would go to—rather, in 1983 it was $27 billion, but it will go to $80 billion in constant dollars in 10 years. These figures come from HCFA projections.

Mr. **Gore**. HCFA, incidentally, is the Health and Financing Administration.

Dr. **Khachaturian**. Now, the actual figures, the amounts that were spent for Alzheimer's disease, I have the figures from 1976 to 1984. This is not the total amount by the Public Health Service. It includes NIA, National Institute on Aging, National Institutes of Mental Health, NINCDS, and NIAID.

In 1976, $3.8 million was spent; in 1977, $4.6; 1978, $5.1; in 1979, $9.6; in 1980—I can't see well—$13; in 1981, $16.7; in 1982, $20.3; in 1983, $25—$26.5; and estimates for 1984 are $36.4 million.

Mr. **Gore**. All right, thank you.

Let me ask you, Dr. Khachaturian: The current issue of Science magazine, which was published only 4 days ago——

Dr. **Khachaturian**. That's right.

Mr. **Gore** [continuing]. Includes a report from two researchers at Harvard Medical School, Dr. Charles Marotta and Dr. Elizabeth Sajdel-Sulkowska, about a new finding that came out of studies of frozen brain cells. I won't go into the details that were reported in the magazine article; but essentially, what they found was that there is a profound shortage of ribonucleic acid—is that correct?—in the brains of Alzheimer's victims?

Dr. **Khachaturian**. That's right.

Mr. **Gore**. And they are inferring from this discovery that there is an RNA-DNA defect at the—there may be a DNA defect at the heart of the disease mechanism with Alzheimer's disease.

How promising do you think this discovery is, and how much hope should we get from it that it will open the door to very promising new lines of research that could lead to a cure for this disease?

Dr. **Khachaturian**. OK, the discovery, essentially, was that this RNA, which is a chemical involved in synthesizing new protein, is considerably less in Alzheimer's brains. The other important finding which relates to the one they reported is that there is an enzyme which is involved in digesting this ribonucleic acid. Once in a while you need to control this synthesis of new protein. What they found is that there is another enzyme which apparently inhibits this other enzyme that controls the RNA. That, then, goes unchecked, which means that it keeps on taking away this enzyme which controls the DNA—rather "R".

Mr. **Gore**. OK, let me try to put that in simple language, and you correct me if I do grave damage to it in the process.

There is DNA and there is RNA. DNA is the code of life.

Dr. **Khachaturian**. Genes.

Mr. **Gore**. It controls the genes, it is the blueprint of life. It has the double helix——

Dr. **Khachaturian**. That's right.

Mr. **Gore** [continuing]. And everyone's heard about it. RNA is something that is produced by DNA. DNA stays inside the cells and it produces an almost exact copy of itself that it sends out to give instructions to other cells.

OK? So far, so good?

Those who are familiar with computers, here, will know that there is a hard memory and then they have the printout, you do a duplicate disk. Well, DNA produces a copy called RNA, that is sent out into the body, and, in this case, into the brain, to give instructions on how to keep the normal operations going.

In this case there is a chemical called an enzyme, which eats up that RNA. The DNA sends it out with instructions, to the brain, to proceed with normal operations; but it runs into this enzyme which acts almost like an acid in that it eats it up. And what they have found is that there is another enzyme that eats up the first enzyme and keeps it all in balance. But in the brains of Alzheimer's patients, that second enzyme that holds down the first one, the one that eats up the RNA, that is the one that is missing; and, so, the first enzyme that eats up this copy—or RNA, the messenger with the instructions—is eaten up, and it doesn't get through. The messenger doesn't get through. It's as if Paul Revere was knocked down before he got to the place where he could issue the warning.

Have I done grave damage to the scientific——

Dr. **Khachaturian**. No—the basic notion.

Mr. **Pepper**. You did a good job. [Applause.]

Dr. **Khachaturian**. I think it's an excellent description, but the end result, I think, for the layman to understand is that this is an extremely significant finding. Because now a lot of the things that I described can be explained by this mechanism; that is, the reduced ability of the cell to produce the chemicals that are needed for communicating, repairing. Because we know that in old age, contrary to our previous beliefs, the nerve cells can grow and form new connections; that is, in old age one could still learn——

Mr. **Gore**. Yes.

Dr. **Khachaturian** [continuing]. The brain can make new connections. What this new finding indicates: that this ability of forming new connections is not possible because the nerve cell cannot produce these new proteins.

Mr. **Gore**. Well, these researchers who have just made this discovery are going to be witnesses in the first public, indepth explanation of their findings at our later hearings in 3 weeks, and we're looking forward to that.

Now, let me ask you another question. There have been some reports about a connection between aluminum and Alzheimer's, and my feeling is that maybe we need to clear the air a little bit about that, because I've heard several people comment on it. And based upon what I've learned about it, there may have been an overstatement of what that connection is.

Again, you correct me if I'm wrong. I'm going to try to state it simply, and you correct me if I'm wrong.

They have found from autopsies a much higher concentration of aluminum in the brains of Alzheimer's victims.

Dr. KHACHATURIAN. Only in cells that have the lesions that I mentioned; that is, in Alzheimer's brains there are some cells that have lesions in them called neurofibrillary tangles.

Mr. GORE. Yes.

Dr. KHACHATURIAN. For short they are called tangles.

Mr. GORE. In those——

Dr. KHACHATURIAN. In those cells there is a high concentration of aluminum.

Mr. GORE. OK. Now, some people jumped to the conclusion that there was a causal—there might be a causal—relationship. But that's probably wrong, isn't it?

Dr. KHACHATURIAN. We don't know whether it's a cause or a consequence of the pathology. It is possible that cells that are damaged by the presence of these tangles are susceptible to getting aluminum. We have to remember that as one ages the amount of aluminum that's in the brain increases.

Mr. GORE. Sure.

Dr. KHACHATURIAN. And aluminum is one of the most abundant substances. It's all over the place.

Mr. GORE. To state that another way, all of us consume aluminum constantly; it's ubiquitous in the environment.

Dr. KHACHATURIAN. Exactly.

Mr. GORE. And there may very well be something about Alzheimer's disease that causes its victims to retain more aluminum, and it may have no causal relationship whatsoever.

Now, let me ask you another question related to this:

Isn't it true that they have done epidemiological studies of people who work with and around aluminum, and they have found there is no higher incidence of Alzheimer's disease, whatsoever, among people who have worked with aluminum all their lives? Isn't that the case?

Dr. KHACHATURIAN. That's true.

Mr. GORE. OK, I wanted to clear that up and get that on the record; but, of course, it's something that bears further study.

Dr. Cook-Deegan, is it fair to say that the current system for reacting to and coping with Alzheimer's disease, if you look at all of the agencies, and all of the medical societies, and everyone involved, there's a kind of an uncoordinated patchwork at the current time?

Dr. COOK-DEEGAN. Yes. And I would not be original in saying that. Yes.

Mr. Gore. It isn't tied together and there's no coherent plan for coping with this problem; is that correct?

Dr. Cook-Deegan. That is absolutely true. And the worst defect in the system is trying to deal with the problem of long-term care.

Mr. Gore. OK. Well, that's really one of the central challenges that we're going to try to face in these 3 days of hearings, is to try to make sense out of that patchwork and come up with recommendations for a coherent policy.

Again, as with the first panel, we could certainly spend a great deal more time with you; but because of the pressures to move on, I'm going to have to ask the next panel to come. But I want to thank both of you for your testimony here. Thank you very much. [Applause.]

Now, let me call the third panel of witnesses: Mr. William M. Stephens with the Tennessee Commission on Aging; Dr. Robert Fox with the Medical Education and Family Practice part of East Tennessee State University; Ms. Eleanor Carper with the University of Tennessee in Memphis; Mrs. Mary M. Lancaster, R.N., with the Veterans' Administration Center at Mountain Home, TN.

While these witnesses are working their way to the witness table, let me make another announcement for our guests here today. You remember that I said we are going to end this hearing at 1 o'clock. We are going to put a signup sheet at the witness table here, maybe a couple of them, for convenience. We've got several signup sheets. Anyone who wishes to receive additional information on Alzheimer's disease, and particularly on how families can better cope with Alzheimer's disease, at the conclusion of the hearing you are welcome to put your name and address on the signup sheet which will be at the witness table. And we will send you information packets perpared by the Congressional Research Service of the Library of Congress, along with other materials that we will assemble for you, including a guide for Alzheimer's patients' families that Senator Pepper and his Aging Committee have put together. So, remember that at the conclusion of the hearing if you wish to put your names and addresses on that signup sheet.

Now, then, we'll go to our third panel.

We are delighted to have you here. The pressure of time is, unfortunately, increasing as each panel comes up, because it is so interesting and so valuable. So, if you could keep your prepared statements short, we would appreciate that. Without objection your full statements will appear in the record. We're very glad to have you here.

We'll begin with Mr. William M. Stephens with the Tennessee Commission on Aging, our State government's agency on aging. And we're delighted to have you here to lead off this panel, Mr. Stephens.

STATEMENT OF WILLIAM M. STEPHENS, DIRECTOR OF LEGAL CARE, TENNESSEE COMMISSION ON AGING, ACCOMPANIED BY DR. ROBERT D. FOX, ASSOCIATE PROFESSOR OF MEDICAL EDUCATION AND FAMILY PRACTICE, EAST TENNESSEE STATE UNIVERSITY; MS. ELEANOR CARPER, M.P.H., UNIVERSITY OF TENNESSEE; AND MRS. MARY M. LANCASTER, R.N., VETERANS' ADMINISTRATION CENTER, MOUNTAIN HOME, TN

Mr. STEPHENS. Thank you very much. I'm delighted to be here.

The commission on aging is doing a great deal, beginning this year, in training State and area agency on aging personnel on the subject of Alzheimer's, with the main intention of developing more support groups and expanding support groups.

Tennessee has some very fine Alzheimer support groups: Earl Caldwell, in Pigeon Forge, who has made Alzheimer's his life since his wife became a victim, has developed a very large support group in east Tennessee, which is now affiliated with the University of Tennessee. Through Earl's outreach efforts support groups are now beginning in the tricities area, as was mentioned earlier. Dot King in Memphis, a R.N. whose sister is an Alzheimer's victim, also has made Alzheimer's her life. In Nashville, Judy Schouland has a very fine support group which is expanding. In Chattanooga, the area agency on aging, Viston Taylor and Marcia Davis started an Alzheimer's group which is expanding—has resulted in one new group being started in Cleveland.

If I had an opportunity to answer the question that Congressman Gore asked the first group—"What would you advise someone who has just learned that a member of the family is a victim of Alzheimer's?"—I would say to get in contact with a support group and to get to work right away on legal problems—legal and financial problems. This area has been touched on briefly.

The financial problems and legal problems are very formidable for some people under the pressure of caring for someone who can't go into a nursing home—perhaps if they want the person to—because nursing homes don't want Alzheimer's patients. This is one of the problems that is quite severe in some areas. They don't know where to turn. They don't think about taking care of the paperwork, the legal work, in time. And if a person has now become totally disabled or incompetent, the only recourse is a petition for guardianship or conservatorship, which requires attorneys' fees, court costs, extensive procedures.

If people, when they first are confronted by this terrible problem, will have a durable power of attorney drawn up, this can go a long way toward helping them. Tennessee passed the durable power of attorney law just last year. It isn't widely known. Basically, of course, an ordinary power of attorney becomes void when the person who gave it becomes incompetent. It requires a power of attorney which is set up specifically to take effect after the person has become incompetent or to continue after that. That can take care of a lot of situations.

Another very important thing is a living will; 21 States have recently passed living will legislation. Tennessee is not one of them; although six of the States bordering Tennessee have passed living will legislation.

A living will, of course, is simply a declaration that one does not want to be kept alive by life-support systems when that person becomes terminally ill, comatose, unable to make decisions. It is one of the few legal instruments that can actually eliminate much suffering; because all of us realize how horrible it will be to be kept alive for weeks, or months, or years, when we're not able to say, "Pull the plug," or the doctor refuses to do it because he is afraid he will be sued for malpractice.

The most enlightened living will legislation provides that doctors are protected so that they, if they do—if they go along with the living will—they will be protected from any kind of legal action.

Senator Pepper mentioned the problems of Medicare—they've been mentioned. I'd like to say just a little about this, because the Medicare legislation, as it was originally written, did a much better job than Medicare is doing today, mostly because the HCFA doesn't follow the law and the regulations in its manual. That's one of the problems.

In 1968, Medicare paid 16 percent of all nursing home costs—16 percent—Medicare. Now it's probably less then 1 percent; the reason being that nursing homes who qualify a patient for skilled care and Medicare are punished by HCFA if they are overruled by the fiscal intermediary. So, the nursing home has to eat the expense, and they learn right—very fast—not to approve, because if they disapprove they're never overruled.

In the State of Tennessee, we have about 72.5 percent, I believe, of nursing home patients that are on Medicaid, but only 3.2 percent are on Medicare. And that says a lot right there. Medicare is insignificant insofar as paying for nursing home care, as has been mentioned before. It would be terrifically expensive, of course, to cover intermediate care, but in some States such as Tennessee there's practically no difference between intermediate care and skilled care. About 95 percent of the nursing home beds in Tennessee are intermediate care; Medicare pays nothing. There are only 139 beds in all of this State that are set aside for Medicare patients; so, it's a formidable problem.

I'd like to just mention briefly two specific cases that I'm very familiar with.

By the way, I'd like to mention that two members of my family, too, have been Alzheimer's patients. Although undiagnosed, I feel they were.

One of the big problems in nursing homes, besides the fact that it's hard to get an Alzheimer's patient in, is that nursing homes discriminate against Medicaid. Now, I have a contract in front of me. I removed the identifying information, because the family of this elderly woman who is an Alzheimer's victim are afraid of recriminations.

She exhausted all of her resources, she was told that she was eligible for Medicaid—her family was told. Her children are in their sixties, and they've been caring for this woman for years. They made the sad decision to put her in a nursing home. They talked to a nursing home administrator who said, "No, don't apply for Medicaid now, because we can't give you a Medicaid bed until you've paid private pay rates for 12 months." And, so, it's typed on the contract: "The undersigned responsible party understands and agrees that the patient cannot be eligible for a Medicaid bed at our facility within one year following admission."

This is a countrywide problem. The Medicaid-Medicare antifraud and abuse statute provides a penalty of $25,000 and a couple of years imprisonment for a nursing home administrator who requires supplementation of Medicaid, who requires this kind of a contract. This contract was made early this year. And I don't know what the Federal Government can do about this, but one thing would be to provide for attorneys' fees for a successful litigant against a nursing home who has deprived a patient of her rights to receive public benefits. This is just as serious a matter as discriminating against people on the basis of race, and sex, and age.

Another case I worked with for about 3 months involves a woman who grew up very close to here at Milligan. Her grandfather was Dr. Minant, a well known country doctor here in the early part of this century. She was injured by—run over by—a truck when she was 6 years old, and since that time she's had extensive medical problems. She's not an Alzheimer's patient, but her problem is typical of Alzheimer's patients.

As of last December, as a disabled widow she was drawing $296 Social Security disability; which made her eligible for SSI, which gave her Medicaid. She got out of a nursing home because she had a very good doctor who was willing to have her try to live independently. She has lived 5 years on Social Security in a retirement home. Because she had Medicare, her oxygen which she requires every 2 hours, was paid for, and she was receiving one visit a week from a home health care agency which did her heavy work.

Then Congress passed a bill as an amendment to the Social Security Act 1983 Amendments, giving a raise to disabled widows. Now, this is a situation where Congress was well-meaning. This raise to disabled widows knocked my friend off Medicaid. She received notice that she was no longer eligible.

She appealed. The Department of Human Services tells her, "No, you cannot receive Medicaid unless you are either pregnant, under 21, or living in a nursing home."

She requires 10 medicines every day. Medicaid paid her medicines. Now, she can still get by. She gets $3 more a month than she got before the raise. Now, as long as she remains healthy, she can manage to make it; although, the medicines cost her $100 a month—which Medicaid used to pay. If she goes into a nursing home, under intermediate care it will cost the State of Tennessee and the Federal Government together about 900 and something dollars; and yet, she is being forced by the bureaucracy to return to a nursing home because she doesn't have Medicaid.

Now, this is an outrageous result from well-meaning legislation. And, as I say, I know it was well intentioned, but it seems to me that Congress when passing any kind of a bill intended to benefit someone, any kind of public benefits, should provide that this cannot operate in any way to hurt someone. It seems to me that this should be done and could be done, and we would strongly urge it. I would strongly urge it, personally, and the commission on aging would as well.

And we want to urge this subcommittee, Congressman Pepper and Congressman Gore, in your legislation, to please don't lose sight of the legal problems in the legislation. But the problem of Patricia Foster, the woman who is being forced back into a nursing home, if she once gets sick she can only go back into a nursing home, she can't pay the $356 Medicaid deductible. There's no way in the world she can pay it.

I urge you very strongly to pass some kind of legislation to help Mrs. Foster and the many other disabled widows, some of whom are Alzheimer's victims. The Alzheimer's victims generally can't speak out for themselves; she can. But a lot have been hurt by this.

Thank you.

[The prepared statement of Mr. Stephens follows:]

ALZHEIMER'S HEARING -- August 30, 1984

William M. Stephens, Director of Legal Care,

Tennessee Commission on Aging

The Administration on Aging has initiated a program to inform the general public about Alzheimer's Disease and to train the staff of state and area agencies on aging. The objective is to help in the formation of additional support groups for Alzheimer's families, therby aiding the patients. In response to this initiative, the Tennessee Commission on Aging will sponsor a statewide workshop in the late fall, with the aim of increasing and expanding Alzheimer's support groups in the state.

Presently the state has large and well-organized support groups in Knoxville, Nashville, Chattanooga, and Memphis, with smaller groups existing in several other areas. Two of the groups are affiliated with the Alzheimer's Disease and Related Disorders Association (ADRDA), and one of these (the Knoxville group, largest in the state) is associated with the University of Tennessee. All groups meet at least once each month and are engaged in extensive outreach and community education.

The Commission on Aging, through its Legal Care and Long-Term Care Ombudsman programs, has assisted the Alzheimer's groups in this state by presenting workshops to inform families of their legal options; by assisting in nursing home placement where appropriate; and by providing legal advice and representation.

A vital need for all Alzheimer's victims and their families is legal planning. They must prepare for the time when the patients will be incompetent--or at least incapacitated. Someone will have to sign consent forms for medical tests and treatment, and to make decisions about virtually every aspect of patients' lives. Someone must have the legal authority to sell or mortgage property, and to write checks. Someone must sign as responsible party for admission to nursing homes and to apply for Medicaid--which is indispensable in most situations as resources are often exhausted by the time institutionalization is required.

For those families who failed to plan ahead (and whose loved ones are already incapable of making decisions), the only legal remedy is a petition for guardianship or conservatorship. These procedures require court hearings, legal fees, and court costs. Often there is no close relative or friend available to serve as guardian or conservator. Because so many have no one to serve in such capacities, we see a growing need for a statewide guardianship program. The Commission on Aging has established a committee which is assessing this problem and surveying guardianship programs in other states. On August 14, 1984, the retiring Commission voted unanimously to recommend that the new Commission work toward the creation of a State Office of Public Guardianship.

The Tennessee Legal Care network (developed by the Commission on Aging with funds provided under the Older Americans Act) has already begun the task of training private lawyers in guardianship procedures and problems. One workshop has already been presented (see attached newsletter "TLC"), and others are being planned.

For persons in the early stages of Alzheimer's, advance planning can obviate the need and expense of guardianship. Through the wise and timely use of wills, trusts, durable powers-of-attorney, the establishment of joint ownership of assets with right of survivorship, and the use of "living wills," the devastating eventualities of Alzheimer's can be softened somewhat and made more manageable.

A new legal instrument that can actually alleviate some of the suffering of Alzheimer's is the "living will." This is a document that formalizes a person's wish to avoid the prolongation of the process of dying. A written document is needed, of course, because of the physician's duty to preserve life. Since so many of us fear and dread the prospect of being kept biologically alive long after brain activity has ceased, a movement to legalize living wills is sweeping the country. Already 21 states (including six bordering Tennessee) have passed right-to-die legislation. In most states that have enacted these laws, doctors and hospitals are given immunity from any lawsuit resulting from their acceptance of a patient's written wish to be allowed to die and not be kept alive by artificial means.

Living wills are particularly important for Alzheimer's patients. If they are able to execute such a document while they are still capable of making informed and rational decisions, families and care-givers alike can benefit in many ways--not only in reducing suffering but in greatly lessening the expense of a protracted illness that seems never-ending. In California and Oregon (two of the first states to adopt right-to-die laws), recent amendments allow the appointment of a proxy to be named within the living will. Such proxy can make the all-important decisions about the continuation or cessation of life-support when the patient is incompetent or *in extremis.*

In 1983 Tennessee enacted a new durable power-of-attorney law, which permits a person to condition a power-of-attorney on the onset of disability, and also to name a guardian or conservator in the instrument itself. Even greater flexibility for Alzheimer's patients would be provided by the enactment of right-to-die legislation. Despite the fact that Tennessee does not legally recognize living wills, many senior citizens are writing their own, or asking their lawyers to draw them up; and we are told that some physicians are accepting them and acceding to their patients' wishes--at least under some circumstances.

Since 1982 the Commission on Aging has issued a newspaper column "Answers on Aging", which now appears in 17 Tennessee papers and three senior citizens publications. On the average we receive from 100 to 200 letters a month from senior citizens posing legal problems (which are referred to Legal Care attorneys or private lawyers). In recent months a great deal of our mail relates to Alzheimer's patients and their problems, including their need for living wills. Some representative columns dealing with such problems are attached, along with a copy of TLC featuring a discussion of living wills.

The Tennessee Legal Care network operates through local, nonprofit organizations which employ their own attorneys and paralegals. The Commission on Aging furnishes funding, support, technical assistance, and training in areas of prime importance to the frail and institutionalized elderly. In addition to guardianship and its alternatives, we have provided training in nursing home law and litigation, Medicare and Medicaid, Social Security and SSI, and age discrimination. Because the legal needs of elderly people are so specialized, and the physical problems of communicating their needs so formidable, special outreach to homes and institutions is necessary. With only 12 Legal Care attorneys in the state, and an elderly population of 765,000, of whom more than 20% are below poverty level, we can provide direct representation to only a fraction of those who need services. Fortunately we have a small but dedicated group of private attorneys who handle many of the cases without any fee at all or for a reduced fee.

I should point out that the Tennessee Legal Care projects cost the taxpayers only about $320,000 a year total. This amounts to less than $1 for each elderly Tennessean. Which is not enough, of course, to do the job we would like to do. Unfortunately, the Older Americans Act, while mandating legal services for the elderly, does not allocate any special funds to it or require that a certain amount be spent on it. Consequently some area agencies on aging give it a very low priority. One of our Legal Care projects receives only $10,000 a year to provide Legal Care throughout nine counties.

I would like to describe two current cases which represent, I am afraid, only the tip of a huge iceberg. These two tragedies illustrate a great need for action on the federal level. The first case deals with Medicaid discrimination--that is, discrimination by nursing homes against persons eligible for Medicaid benefits. If it is true that 50 to 60% of nursing home patients are victims of Alzheimer's (as has been reported by the National Institutes on Health), the problem of Medicaid discrimination is a major one for families of Alzheimer's patients. In this first case I am not using the patient's real name, since her family fears retaliation by the nursing home.

Bessie Smith is a widow of 84 who exhibits Alzheimer's symptoms. Until recently she was cared for by her son and daughter, who are 62 and 60 years old respectively and have families of their own. After Mrs. Smith became totally unmanageable, her son and daughter had no choice but to place her in a nursing home. Her resources were now exhausted and they were informed by the Department of Human Services that she would qualify for Medicaid. However, the nursing home administrator told them they should hold off applying for Medicaid. They would have to pay private-pay rates for 12 months, he said, before a Medicaid bed could be made available.

Consequently, they signed a contract agreeing to pay $40 a day for a year, which they could pay only at considerable hardship. Two months later they read a column I had written about Medicaid discrimination and telephoned me. Subsequently they gave me a copy of the illegal contract they had been required to sign, and it is attached hereto. At their request we have eliminated identifying information. The following words, typed on the form, are quite legible:

> "The undersigned responsible party understands and agrees that the patient cannot be eligible for a Medicaid bed at our facility within (1) one year following admission."

Under federal law [42 USCA 1396h(d)(2)] a nursing home administrator who charges a Medicaid-eligible patient over and above what Medicaid allows is guilty of a felony and may be imprisoned for five years and fined $25,000. Although the law is quite clear, and many nursing homes throughout the country are in violation, we are informed by the National Senior Citizens Law Center that no nursing home administrator has ever been prosecuted under this statute. Some nursing homes have been held civilly liable, however, in state courts, for preventing persons from receiving state and federal benefits to which they are legally entitled.

In the other case I would like to describe, the victim (not of Alzheimer's but of a bureaucratic Catch-22) has given me permission to use her name. She is Patricia K. Foster of Nashville. A disabled widow caught in the same unfortunate situation that some Alzheimer's victims finds themselves in, Patricia Foster is an articulate and courageous woman who grew up in Milligan, only a few miles from the campus here at East Tennessee State University.

When Patti Foster was six years old, she was run over by a truck which crushed almost every bone in her body. Since that time, she has rarely gone six months without being hospitalized and has had more than a dozen major operations. Despite this, she has led a full life and ran her own business for years. After her husband died of a heart attack and she was left destitute, she managed to raise her only daughter and get her through school before she herself collasped and was put into a nursing home. After some time there, she realized she had to get out because, as she put it, she was surrounded by people who had given up and she was not about to give up yet. Fortunately, Mrs. Foster's doctor agreed that she should attempt to live independently and he found a retirement home for her. Despite her many physical problems, she has done well for five years, since Medicare pays for the oxygen she needs every two hours and Medicaid buys seven of her daily medications. As of last December, her only income consisted of a Social Security disability check of $296 per month plus $38 SSI.

Then Congress passed a raise for disabled widows. Patricia Foster received an increase of $52 which put her over the SSI ceiling and caused her to lose her Medicaid. She appealed, but the appeal was denied and she was told that she could receive Medicaid only if she was (1) pregnant, (2) under 21, or (3) living in a nursing home. So, because of a raise which put only $3 more in her pocket each month, she has lost her Medicaid and the system seems determined to force her back into a nursing home.

Before she goes back, she says, she will jump off the top of the Metro Teachers Apartment, where she lives. I don't believe she will, because she is too courageous to give up, but that's what she says.

She has begun a letter-writing campaign and she is still holding on. If she makes it until October 22, she will set a new record of two years without hospitalization.

Patricia Foster is not the only disabled widow who was hurt by the well-intentioned raise voted by Congress. It seems to me that Congress could prevent this kind of unforseen result by providing that, whenever any kind of raise in benefits is enacted, the beneficiary should be given the option to accept or reject the increase; or by providing that no increase should be allowed to result in a decrease in benefits. In the meantime, I submit, Congress should pass immediate legislation to reinstate to the Medicaid rolls all disabled widows such as Patricia Foster who lost their coverage because of the raise.

Congress could also help many Alzheimer's victims and their families by specifically providing for attorneys' fees in successful suits against nursing homes that have prevented persons such as Bessie Smith from receiving government benefits to which they are entitled. Nursing homes requiring such unlawful contracts are fully as guilty of unlawful discrimination as if they had systematically excluded persons on the basis of race, sex or age.

Another area where federal action is badly needed is in Medicare. I am not speaking of the well-known problems with Medicare Part B, which hospitals themselves could essentially solve by simply withholding staff privileges from physicians who refuse to accept Medicare assignment. I am more concerned by the total lack of Medicare coverage for nursing home residents classified as needing only ICF (Intermediate, or custodial) care, as opposed to Skilled (SNF) care. As families of Alzheimer's patients learn, to their bewilderment, victims of Alzheimer's are generally considered to need only custodial care, which means that Medicare will pay nothing, whether the victim resides in a nursing home or is the recipient of home health care services. Consequently, the burden and the expense must be borne entirely by the families, until all resources have been exhausted and they can apply for Medicaid.

In Tennessee the problem is worse than in most states, for over 90% of our nursing home beds are classified as ICF, or custodial. According to the Tennessee Health Care Association, there are presently 28,025 licensed beds in the state. Of the total number, as of March 16, 1984, 75.2% were Medicaid-certified, while only 3.2% were occupied by patients receiving Medicare. Obviously, Medicare coverage in nursing homes is almost non-existent in this state. It is a cruel blow to families who have broken their hearts, exhausted their bodies, and spent their life savings to care for a loved one day and night, to learn that Medicare cannot help because the patient is entitled only to custodial, and not skilled, care.

Thank you for allowing me to testify at this hearing. Attached to my statement are copies of the Commission on Aging newsletter TLC and several newspaper columns dealing with the topics touched on here.

Biographical Sketch: WILLIAM M. STEPHENS

A seventh-generation Tennessean, William M. Stephens was born in Chattanooga and served overseas with the U.S. Marines during World War II. He graduated from the University of Tennessee law school in 1950 and later received his LL.M. (in Ocean and Coastal Law, a combined marine science and law degree) at the University of Miami. He has taught college courses in law, oceanography, and environmental education, and is the author of three published books (plus a series of children's books) about science and the sea.

Since 1976 Mr. Stephens has specialized in the legal problems of senior citizens. Currently he is Director of Legal Care for the Tennessee Commission on Aging and coordinates the work of ten senior citizen law projects which provide legal assistance to older Tennesseans in all 95 counties. Mr. Stephens writes a newspaper column, "Answers on Aging," which appears in 17 Tennessee newspapers and three senior citizens' publications.

B-14 Nashville Banner, Wednesday, January 4, 1984

Alzheimer's groups offer aid

Q. My husband was a carpenter for 43 years. Now, four years after his retirement, he can't even drive a nail. The first hint of a problem was when he would lose his tools or the keys to his truck. Soon it was happening every day, and half the time he didn't even know my name. Then things got worse. Now he wanders away and a neighbor has to bring him home.

Finally the doctor has put a name on his disease — Alzheimer's. How can I learn more about it and about others who are having to cope with the same tragedy as mine?

A. Alzheimer's disease (also known as senile dementia) is caused by the gradual degeneration of nerve cells in the brain. It is irreversible and there is no known cure. According to the Alzheimer's Disease and Related Disorders Association (ADRDA), it is the third leading cause of death in older people, though often it is not diagnosed. Since Alzheimer's is still not well-known, many of its victims and their families are helpless in their attempts to learn more about the syndrome and where to go for help. Only a few years ago, we are told, some insurance companies refused to pay claims to Alzheimer's victims on the ground that they did not consider Alzheimer's a disabling disease.

BILL STEPHENS

Answers on aging

Tennessee has several Alzheimer's support groups. The largest is the Knox Area chapter of ADRDA, P.O. Box 1100, Pigeon Forge, TN 37963, telephone (615) 453-6466. This group was begun by Earl Caldwell, whose wife has gradually deteriorated over the years and now has assumed a fetal posture with no communication to the outside world. Caldwell presently has 300 families in his group and will furnish information about Alzheimer's to anyone who writes or telephones him. Alzheimer groups also have been started in Nashville and Chattanooga.

Q. After 41 years of marriage, my husband, Wally, died in 1972 and left me a lifetime right to a 200-acre farm. His will stated that I would lose my right to the property if I married again. In 1979, after seven years of widowhood, I married again. Jim, my present husband, and I settled down to live in the house.

First thing we knew, Wally's daughter by his first marriage served a court order and evicted me from my home. A lawyer advised me to sign it over to them. He said I didn't have a leg to stand on and I would just have a lot of expense.

I read in your column that a surviving spouse can dissent from the will and receive one-third. Shouldn't that apply to me?

A. Under the present law in Tennessee, which went into effect in 1976, a surviving spouse who dissents from the will is entitled to one-third of all personal property and real estate. Unfortunately, that law does not apply to you since your husband died in 1972.

Under the old common law, in effect at the time Wally died, a widow was entitled only to her rights of dower, which amounted to a lifetime estate in one-third of the property.

I am wondering what rights you signed over to your husband's daughter on the advice of counsel. If you waived your rights of dower, you may have no claim at this time. If you did not waive your rights, you may still have an interest in the property. I am sending you the name of the Legal Care attorney in your area. I suggest you consult him as soon as possible. If he feels you have a claim, he will refer you to a competent private attorney who can handle it for you.

Attorney William M. Stephens is Director of Legal Care for Tennessee. Send questions to Mr. Stephens at the Tennessee Commission on Aging, 700 Tennessee Bldg., Nashville 37219.

The Knoxville News-Sentinel, Sunday, April 29, 1984 E5

'Private Pay' Illegal

William Stephens

Answers on Aging

Q. I recently put my 87-year-old mother in a nursing home. Even though she has no assets and is eligible for Medicaid, the administrator of the home would not take her unless I signed a contract agreeing to pay "private pay" rates of $45 a day for 18 months. After 18 months, they will accept her as a Medicaid patient and I won't have to pay any more. Is this legal?

My wife and I are not wealthy and we are on Social Security ourselves. If I stop paying, can he put my mother out?

A. Not without violating her rights and leaving himself open to both civil and criminal penalties. According to federal law, a nursing home administrator who requires a Medicaid-eigible patient (or her family) to sign such a contract is guilty of a felony and can be fined $25,000 and imprisoned for five years. This law is found in Vol. 42 of the U.S. Code at Section 139h(d)(2). If you wish to file a complaint, contact the U.S. attorney's office.

Besides it being a crime to require supplementation of Medicaid, such a contract is unenforceable at law. In a case recently decided in New York, the courts said: "Plantiff's apparent intent of denying a poor person the right to make application for assistance which the state and federal government have seen fit to provide cannot be condoned legally or morally."

In New Jersey, a recent study showed that about 1,800 Medicaid-eligible patients in that state were being required to pay private rates. A bill has been filed in the New Jersey legislature in an effort to stop this illegal practice.

I am giving you the names and telephone numbers of both the Legal Care attorney and the nursing home ombudsman in your area. They can advise you and will be of great help if the nursing home brings suit to collect on the contract or attempts reprisals against your mother.

Q: My husband is 78 and terminally ill. He wants to die here at home, where I can be with him constantly. While I would prefer this, too, I am nervous about it. There are times I may need help or special drugs. We have read that Medicare now covers hospice care for the dying. Where can we get details?

A: Yes, hospice care is now covered by Medicare and it pays nearly everything. Since your husband is eligible for Medicare, he could qualify for hospice care if his physician rates his life expectancy at six months or less.

Hospice care is provided in your own home and includes physician and nursing services, drugs and biologicals, counseling, physical therapy and the help of a home health aide. These services are not provided continuously, you understand, but on an intermittent basis as needed. Twenty-four hour care is available only under extreme circumstances.

Each Medicare recipient is eligible for a maximum of 210 lifetime days of hospice care. If the patient never requires hospitalization or "respite" care, the only expense is five percent of the charge for each prescription or $5, whichever is less.

William M. Stephens, lawyer, is director of Legal Care Projects in Tennessee. Address questions to him at the Tennessee Commission on Aging, 700 Tennessee Building, 535 Church St., Nashville, Tenn. 37219.

Nashville Banner, Wednesday, May 23, 1984

Disabled woman gets caught in Social Security Catch-22

BILL STEPHENS

Answers on aging

Q. Although I am totally disabled and need oxygen eight or nine times a day, I have lived alone quite well with the assistance of a weekly visit from a home health homemaker. With my Social Security check of $296 and an SSI check of $38, I have gotten by, since Medicaid paid for seven of my prescriptions.

But, in March, Social Security informed me that, effective April 1, I would receive an increase of $56 a month. (This was a raise voted by Congress to help disabled widows.) Good news? Not exactly. The raise made me over-income for SSI. And, without SSI, I was no longer eligible for Medicaid! The Department of Human Services explained that I could get Medicaid only if I were (1) pregnant, (2) under 21, or (3) living in a nursing home. I can't very well be pregnant at the age of 60, so it leaves me only one option — to spend my remaining days in a nursing home.

Mr. Stephens, please explain to me why the State of Tennessee can be so short-sighted as to force people like me into a nursing home when I can live at home with a minimum of assistance? I will cost the state ten times as much in a nursing home as I would living here in my own place. But, without Medicaid, I cannot stay here. I cannot afford to buy my medications, and if I ever needed hospitalization, they wouldn't take me without a Medicaid card.

Before Congress did me the big favor, I had a total monthly income of $334. Now my total income is $337 . . . but I've lost my Medicaid.

A. I have explored every avenue I know of without success. There seems to be no legal remedy for your plight, and no social or community group that can help. In my opinion, it will require action either by the federal government or the individual states to help you and other disabled widows who have been grievously hurt by the well-intentioned raise voted by Congress. What Congress has done, it can rectify. When I talked with Congressman Bill Boner's office, I was told that the Congressman is aware of the problem, but that relief is unlikely in an election year.

Until two years ago, when budget cuts slashed the Tennessee Medicaid program, people with your kind of problem were protected by the 'medically needy' provisions of the Medicaid law. This would have allowed you to deduct your medical bills from your income in order to meet eligibility criteria. But under current State regulations, senior citizens are excluded from the 'medically needy' category. As you were told by the Department of Human Services, you will now have to go into a nursing home to become eligible for Medicaid.

Q. We read with interest your item about Social Security 'notch years.' I contacted my Congressman about it and he said there were no facts behind it. I need some concrete information so I can put pressure on the Congressman. I also want to present this issue to the Tennessee Republican Party at the next meeting of the Executive Committee, of which I am a member.

A. The 'notch years' were created by Public Law 95-216, enacted by Congress on December 20, 1977, as an amendment to the Social Security Act. This law is found in Title 42 of the U.S. Code Annotated, at Section 403, titled 'Reduction of Insurance Benefits.'

Congressman Bill Boner has introduced legislation to eliminate the inequities caused by the 'notch years' statute.

Attorney William M. Stephens is Director of Legal Care Projects in Tennessee. Send questions to Mr. Stephens at the Tennessee Commission on Aging, 700 Tennessee Building, 535 Church St., Nashville 37219.

B-8 Nashville Banner, Wednesday, June 27, 1984

Woman praises 'dignity' of allowing living wills

BILL STEPHENS

Answers on aging

Q. Thank you for writing about 'living wills.' My husband and I are well-seasoned senior citizens who feel that society has a duty to allow the terminally ill to pass on with dignity.

We have had our living wills written up and notarized, and we hope the state of Tennessee will pass a law legalizing them. We wish to have no part of hospitals or doctors playing God with our bodies, prolonging life with their man-made machines, getting rich on our pain and the emotional trauma of our loved ones, bankrupting our security and depriving our offspring of their inheritance.

A. Since my last mention of living wills in this column, I have learned that five more states have recently passed right-to-die laws, bringing the total to 21 including the District of Columbia. Interestingly, six of the 21 states now having right-to-die laws are contiguous to Tennessee. They are: Alabama, Arkansas, Georgia, Mississippi, North Carolina and Virginia.

In Georgia, the right-to-die proponents were aided by a special visit by Abigail Van Buren, who writes the popular *Dear Abby* column. Ms. Van Buren has recommended living wills to her readers on several occasions and has been honored for her efforts by the Society for the Right to Die. After both houses of the Georgia legislature passed the living will law but the governor had not said whether he would sign it or veto it, "Abby" visited the State Capitol and talked with Governor Joe Frank Harris, who then approved the bill.

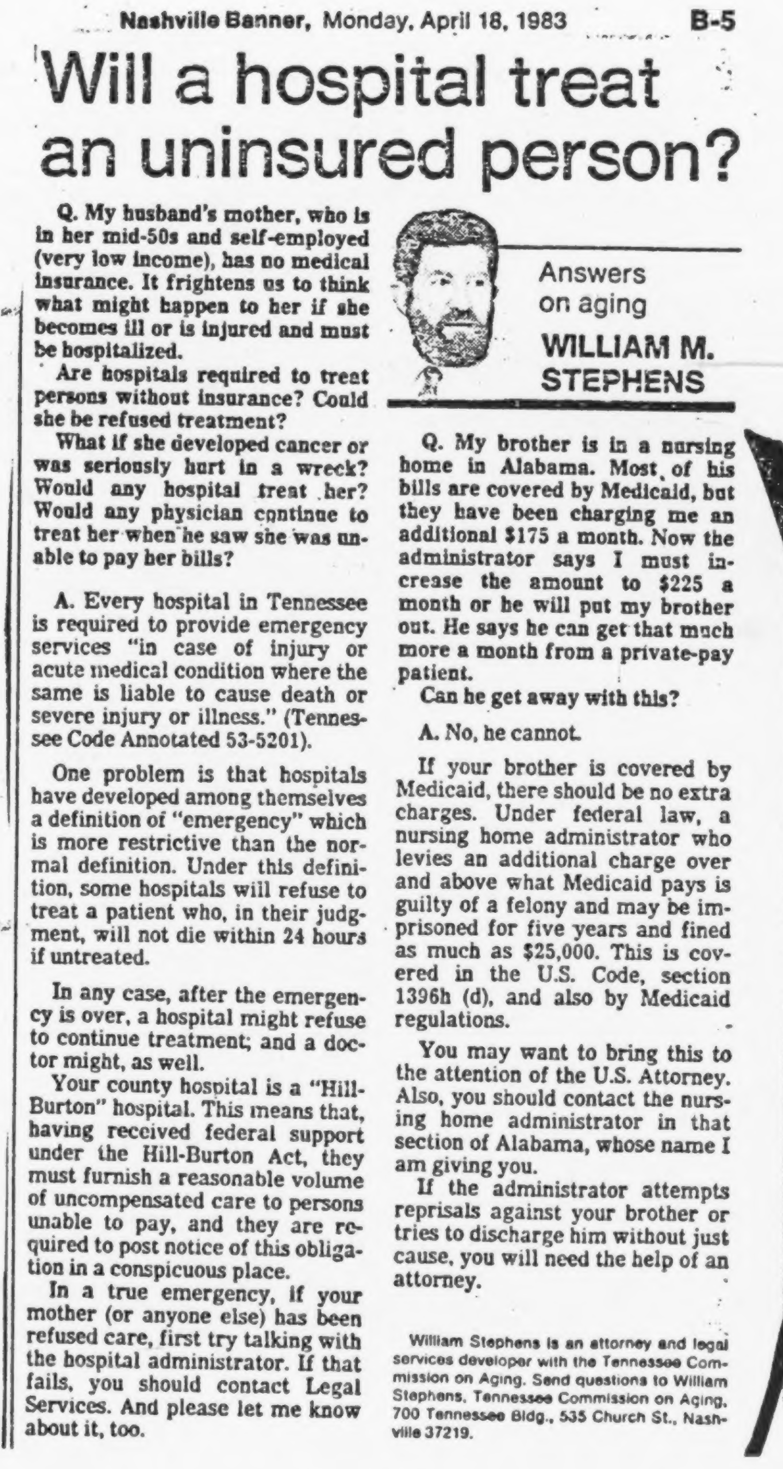
Q. I talked with my lawyer about living wills and he would like more information.

A. Have him get in touch with me. He also should write to: Society for the Right to Die, 250 West 57th St., New York NY 10107. The Society has a wealth of information about right-to-die laws including legal forms and model legislation.

Q. Will life insurance benefits be affected by a living will?

A. No. Although there has been no court test of a living will, it is inconceivable that such a document could be construed as an intention to commit suicide. The execution of a living will should have no effect on insurance.

Attorney William M. Stephens is Director of Legal Care Projects in Tennessee. Send questions to Mr. Stephens at the Tennessee Commission on Aging, 700 Tennessee Building, 535 Church St., Nashville 37219.



Nashville Banner, Monday, April 18, 1983 B-5

Will a hospital treat an uninsured person?

Answers on aging

WILLIAM M. STEPHENS

Q. My husband's mother, who is in her mid-50s and self-employed (very low income), has no medical insurance. It frightens us to think what might happen to her if she becomes ill or is injured and must be hospitalized.

Are hospitals required to treat persons without insurance? Could she be refused treatment?

What if she developed cancer or was seriously hurt in a wreck? Would any hospital treat her? Would any physician continue to treat her when he saw she was unable to pay her bills?

A. Every hospital in Tennessee is required to provide emergency services "in case of injury or acute medical condition where the same is liable to cause death or severe injury or illness." (Tennessee Code Annotated 53-5201).

One problem is that hospitals have developed among themselves a definition of "emergency" which is more restrictive than the normal definition. Under this definition, some hospitals will refuse to treat a patient who, in their judgment, will not die within 24 hours if untreated.

In any case, after the emergency is over, a hospital might refuse to continue treatment; and a doctor might, as well.

Your county hospital is a "Hill-Burton" hospital. This means that, having received federal support under the Hill-Burton Act, they must furnish a reasonable volume of uncompensated care to persons unable to pay, and they are required to post notice of this obligation in a conspicuous place.

In a true emergency, if your mother (or anyone else) has been refused care, first try talking with the hospital administrator. If that fails, you should contact Legal Services. And please let me know about it, too.

Q. My brother is in a nursing home in Alabama. Most of his bills are covered by Medicaid, but they have been charging me an additional $175 a month. Now the administrator says I must increase the amount to $225 a month or he will put my brother out. He says he can get that much more a month from a private-pay patient.

Can he get away with this?

A. No, he cannot.

If your brother is covered by Medicaid, there should be no extra charges. Under federal law, a nursing home administrator who levies an additional charge over and above what Medicaid pays is guilty of a felony and may be imprisoned for five years and fined as much as $25,000. This is covered in the U.S. Code, section 1396h (d), and also by Medicaid regulations.

You may want to bring this to the attention of the U.S. Attorney. Also, you should contact the nursing home administrator in that section of Alabama, whose name I am giving you.

If the administrator attempts reprisals against your brother or tries to discharge him without just cause, you will need the help of an attorney.

William Stephens is an attorney and legal services developer with the Tennessee Commission on Aging. Send questions to William Stephens, Tennessee Commission on Aging, 700 Tennessee Bldg., 535 Church St., Nashville 37219.

B-24 Nashville Banner, Wednesday, September 28, 1983

In Tennessee most people don't have to retire at 70

Q. I understand that the age of forced retirement has been raised to 70. Does this apply to the State of Tennessee as well as to private employers? Please do not use my name.

A. For most employees of the State of Tennessee, mandatory retirement was abolished on July 1, 1979. In other words, there is no upper age limit. You cannot be required to retire on the basis of age.

The only exceptions are state policemen, wildlife officers, firemen, and employees of state universities and community colleges.

Q. Even though I am not yet 65, I am on Medicare because of disability, which is caused by systemic lupus erythema-tosus.

I receive a Social Security Disability check of $480 a month, which is my only income. My doctor and drug bills come to about $125 a month, and my rent is $285. This leaves $70 for groceries, electricity, water, and everything else. I have no savings and my car is 18 years old. After a great deal of time and effort, I managed to get my food stamp allotment increased from $10 to $60 a month.

So here I sit, watching my health deteriorate because I cannot buy all the medicines I need and cannot afford supplemental insurance. I worry constantly about needing to go into a hospital, but being turned down for lack of insurance.

I am told that I am too rich for SSI or Medicaid. I turn to you because I don't know where else to turn.

Answers on aging

WILLIAM M. STEPHENS

A. The only solace I can offer is the assurance that you will not be turned down for hospitalization due to lack of private insurance or a Medicare supplement policy. Things may be harder next year after the Medicare delivery system is changed, but as for now, most hospitals receive Medicare patients with open arms. Medicare pays the entire hospital bill for the first 60 days, with the exception of a deductible of $304, which the patient is supposed to pay.

Your income, unfortunately, is over the SSI edicaid eligibility level, which is $304.30 effective July 1, 1983.

Q. I saw a television report about the government trying to collect Medicaid expenses from children of nursing home patients.

Don't they realize that most nursing home patients are in their 80's, and their children are often drawing Social Security?

My father is 84 and was a much-respected university professor for more than thirty years. He took early retirement because of memory lapses which turned out to be only the first stage of Alzheimer's disease. We cared for him in our home for seven years, until finally we had to give up and put him in a nursing home. He was like a little child who would run away to visit his mother on the farm. (His mother had been dead for 40 years.) During the next five years, we secured the best nursing care money can buy while Dad slowly deteriorated and exhausted all his resources and most of ours. Finally, we were forced to put him on Medicaid.

How in the name of decency and common sense can we be expected to supplement my father's Medicaid? We've already practically put ourselves in the poorhouse.

Worst of all, my husband is showing signs of Alzeimer's too. I don't know how I can possibly care for him at my age if he follows the same pattern as my father.

A. A storm of protest has swept the country in the wake of the federal government's change in its Medicaid manual, intended to allow states for the first time to charge relatives for Medicaid care. Previously, the government has prohibited states from attempting to recoup Medicaid costs from children or relatives of nursing home residents.

Questions have been raised about the procedures followed. Can the government overrule regulations, statutes, and caselaw by changing a manual? Do states have the power to impose such liability on adult children? How could such a program be administered in a fair and equitable way?

Tennessee is one of the few states that has a relative responsibility law on the books, but the applicability of the law is questionable. The Commission on Aging asked the State Attorney General for a legal opinion in the matter. According to Attorney General William M. Leech, Jr., the Tennessee relative responsibility law cannot be enforced against children or guardians of Medicaid recipients. Only parents or spouses of patients could be required to make reimbursements to the state.

Q. Should a will be made in the state in which a person lives? I moved to Tennessee last year but my farm is in Kansas and my will was made in Kansas.

Should I make a new will in Tennessee? I plan to live here the rest of my life.

A. A will must be probated in the state and county where the testator dies. If your will was properly drawn and signed in Kansas in accordance with Kansas law, it may be probated in Tennessee just as if it were made here. After probate in Tennessee, the will may be offered for probate in any other state where you own real property.

Because of the importance of making sure your will operates according to your wishes, it might be wise for you to have the advice of a Tennessee lawyer who is knowledgeable in wills and estates. I have given you the names of several in your community who have told us they will handle cases for senior citizens on a reduced fee basis.

Answers on aging is written by William Stephens, an attorney and legal services developer with the Tennessee Commission on Aging. Send questions to William Stephens, Tennessee Commission on Aging, 700 Tennessee Building, 535 Church St., Nashville 37219.

ADMISSION AGREEMENT

The Life Care [illegible] and [illegible]
(NAME OF NURSING CENTER) (NAME OF PATIENT OR RESPONSIBLE PARTY)

hereby agree to the following terms regarding the health care and financial arrangements pertaining to:

RECEIVED
JUN 7 1984
COMMISSION ON AGING

(NAME OF PATIENT)

THE NURSING CENTER AGREES TO

1. Admit patient upon physician's order and the execution of this agreement or with provisions made therefor, and to assist in obtaining the services of [illegible] and to obtain emergency physician services when required.
2. Maintain written records of all financial transactions with the patient or other responsible party, including records of funds or personal property held in trust for the patient, provided, however, the patient or responsible party requests in writing that the nursing center hold funds or personal property in trust and provided they give the nursing center a written inventory in connection therewith.
3. Furnish room, board, nursing care, and such services as may be required for the health, safety, and well-being of the patient. However, the NURSING CENTER is in no sense an insurer of the safety or welfare and assumes no liability as such.
4. Process required forms for reimbursement for private or public insurance or welfare funds for the patient, if eligible, and apply these funds as they are received against the cost of care. Assist in claims for private insurance benefits, but not to accept assignment thereof unless otherwise noted as an exception herein.
5. Provide required assistance in daily living. Should supportive equipment such as wheelchair, walker, etc. be prescribed for sole, continued use or desired for such by patient, responsible party, it must be supplied by patient or be subject to additional charge.
6. Arrange for the prompt transfer of the patient to a hospital upon physician order, immediately notifying responsible party or next of kin of same.
7. Obtain and administer such medications as may be prescribed, the cost of which will be paid by the patient or responsible party, as billed by the nursing center or direct from pharmacy.
8. Provide an activities program appropriate to the patient's needs and the nursing center's facilities.
9. Furnish bed linens as required for the proper care of the patient under normal conditions.
10. Not knowingly admit or retain a patient who would be of danger to himself or others or whose behavior would be so unacceptable or disturbing as to interfere with the care of others.

THE PATIENT OR RESPONSIBLE PARTY AGREES TO

1. Provide complete information regarding the patient as requested and to authorize release of necessary information to appropriate agencies if requested.
2. Provide a report of negative chest x-ray on admission of patient or within required time limitation.
3. Pay all of the fees and charges described in the contract upon the terms agreed to.
4. Provide or be responsible for personal items of clothing, toiletries, etc. and to make available spending money as required.
5. Discharge the patient from the nursing center upon a five day advance notice.
6. Notify the nursing center at least 30 days in advance of the discharge of the patient not as the result of an emergency.
7. Accept full responsibility for and absolve the nursing center, its personnel and attending physician of responsibility for any event, accident, or deterioration of medical condition while the patient is away from the nursing center and not under the direct care and supervision of the nursing center, or if the patient should leave the nursing center for any reason without first giving notice.
8. Authorize nursing center to effect patient move from room to room for reasons involving nursing, compatibility and the convenience of the nursing center, within the type of accommodation set forth in this agreement.
9. Abide by the nursing center's policies and regulations regarding visiting hours, the patient, and conduct of nursing center operations.
10. Pay all costs, expenses and reasonable attorney's fees, whether suit be brought, in the event same must be expended in the collection of any and all sums due and owing by the patient and responsible party to the NURSING CENTER.
11. Abide by the medication distribution policies and procedures, including the use of a unit dose system which requires that all medication be supplied to conform with the system in use.

FINANCIAL AGREEMENT

The patient or responsible party agrees to pay monthly, and the nursing center will accept this arrangement in full consideration for care and services rendered as follows:

1. Room, board, laundered linens and bedding, nursing care, personal services and such services required for the health, safety and well-being of the patient in accommodation type of 2-bed ICF for a basic fee of $ 40.00 per day. Patient or responsible party agrees to pay all fees and charges not covered by insurance or public aid funds for any reason, when they are billed and thereafter in the event insurance and/or public aid funds are not available.
   - (a) The basic rate includes room, board, and nursing/personal care.
   - (b) Extra charges include personal laundry, special supplies, house medications, prescription drugs, laboratory and therapy fees, personal nursing care equipment rental, physician and private duty nursing fees, ambulance transportation, and personal toiletries/items.

   The daily rate is charged for the day of admission and there is no day charge for the day of discharge if it is by 11:00 a.m.
   The daily rate will be charged on the day of death if applicable.
   A payment equal to 30 days routine care and services will be required upon admission.
   Daily rate changes may be made at any time upon written notification.
   The refund of any account balance will be made within 30 days following discharge or payment in full by all public aid funds. $______
2. Allowance for spending money $______
3. Other (Itemize): $______

Sub Total . . $______

4. The financial cost agreement with the patient, public assistance department or responsible party if patient receives public aid is as follows: $______

Total Cost . . $______

5. ☐ The services for any bill rendered by the physician will be charged to the patient.
   ☐ The services of the physician will be billed by him directly to the patient or responsible party.
6. ☐ Special medications as ordered by the physician from the pharmacist will be charged to the patient.
   ☐ Special medications as ordered by the physician will be billed by the pharmacist directly to the patient or responsible party.

DURATION OF AGREEMENT

Either party may terminate this agreement on 30 days written notice. Otherwise, it will remain in effect until discharge of the patient.

EXCEPTIONS

The parties agree to the following exceptions or additions to this agreement:

The undersigned responsible party understands and agrees that the patient cannot be eligible for a medicaid bed at our facility within (1) one year following admission.

RECORD OF AMENDMENTS TO AGREEMENT

See attached

ENTIRE AGREEMENT

This agreement, together with the patient's admitting record, constitute the entire agreement between the parties, and it may not be amended except by written agreement of the parties.

______________________   /1/84   X ______________________
(SIGNATURE OF NURSING CENTER REPRESENTATIVE)   DATE   (SIGNATURE OF PATIENT OR RESPONSIBLE PARTY)

______________________   ______________________   ______________________
WITNESS   DATE   RELATIONSHIP TO PATIENT

Before me, the undersigned authority, personally appeared ______________________ the sponsor of the patient, who acknowledged before me that he/she executed the same and foregoing instrument as the "responsible party" for the purposes therein set forth.

______________________ Dated this ____ day of ______________, 19 ______________________
WITNESS SIGNATURE   NOTARY PUBLIC

TENNESSEE LEGAL CARE

Vol. 3, No. 3, Summer 1984

LIVING WILLS

The "living will" concept is snowballing, sparked by the Society for the Right to Die, the AARP, the Gray Panthers, and millions of senior citizens. As Maggie Kuhn, founder of the Gray Panthers, recently put it, "When you're hooked up to a machine, it's an affront. Most people in their right minds would want to die quietly."

During 1984 five states have enacted "living will" legislation, bringing the number of jurisdictions with "right-do-die" laws up to 21 including the district of Columbia. The new states with living will legislation are Georgia, Mississippi, West Virginia, Wisconsin, and Wyoming. It is interesting to note that six of the states that have legalized this important new concept are states which border Tennessee. These are Alabama, Arkansas, Georgia, Mississippi, North Carolina, and Virginia. So far, however, no living will legislation has been introduced in Tennessee.

Recently, Wyoming's legal services developer, Donna Rice McCrea, gave TLC a blow-by-blow description of how the legislation passed in Wyoming after heated debates in the legislature fueled by grass-roots support from senior citizens. During the debate, older people throughout the state telephoned a toll-free number at the Commission on Aging to voice their support for living wills.

Ms. McCrea reports that the major resistance to the Wyoming bill came from "right-to-life" groups. Earlier, in Colorado, the medical profession defeated similar legislation because it provided for penalties to be imposed on doctors. Wyoming had time to react to the Colorado failure and consequently removed all penalties against physicians prior to introduction of the bill. Under the Wyoming law, a doctor who refuses to honor a living will has an obligation to transfer the patient to another doctor. Because of this change, the Wyoming Medical Association supported the legislation, and it passed.

"The bill was amended in another way that disturbs me, however," stated Ms. McCrea. "As it finally passed, the providing of nourishment by whatever means is not considered to be the artificial prolonging of life. To me and many others, artificial feeding *is* the ar-

LIVING WILL DECLARATION

To My Family, Physician and Medical Facility

I, ______________________________, being of sound mind, voluntarily make known my desire that my dying shall not be artificially prolonged under the following circumstances:

If I should have an injury, disease or illness regarded by my physician as incurable and terminal, and if my physician determines that the application of life-sustaining procedures would serve only to prolong artificially the dying process, I direct that such procedures be withheld or withdrawn and that I be permitted to die. I want treatment limited to those measures that will provide me with maximum comfort and freedom from pain. Should I become unable to participate in decisions with respect to my medical treatment, it is my intention that these directions be honored by my family and physician(s) as a final expression of my legal right to refuse medical treatment, and I accept the consequences of this refusal.

Signed______________________________ Date____________________

Witness ____________________ Witness ____________________

DESIGNATION CLAUSE (optional*)

Should I become comatose, incompetent or otherwise mentally or physically incapable of communication, I authorize ______________________________,

presently residing at ______________________________

to make treatment decisions on my behalf in accordance with my Living Will Declaration. I have discussed my wishes concerning terminal care with this person, and I trust his/her judgment on my behalf.

Signed______________________________ Date____________________

Witness ____________________ Witness ____________________

*If I have not designated a proxy as provided above, I understand that my Living Will Declaration shall nevertheless be given effect should the appropriate circumstances arise.

tificial prolongation of life. Next year we may offer an amendment to remove this impediment."

Georgia's living will law, which went into effect on July 1, was helped markedly by Abigail Van Buren, who writes the widely syndicated "Dear Abby" column. Several "Dear Abby" columns have mentioned living wills, and Ms. Van Buren has been honored by the Society for the Right to Die for her strong stand on the subject. After both houses of the Georgia legislature passed the new law but the Governor had not signed it, Abigail Van Buren visited the State Capitol and met with Governor Joe Frank Harris. After meeting with "Abby," the Governor approved the bill.

So what exactly is a living will? It is a document that formalizes one's wish to avoid the prolongation of the process of dying. A written document is needed because a physician has a duty to preserve life. Modern technology makes it possible to keep a patient's heart pumping long after brain activity has ceased. The idea of using artificial means to keep a person biologically alive for a prolonged period is abhorrent to many of us. We feel we should have the right to make important decisions about life and death while our health is good and our mind clear.

In most states that have enacted right-to-die laws, doctors and hospitals are given immunity from any lawsuit resulting from their acceptance of a patient's wish to be allowed to die. In several states, however, the living will is not binding on physicians and they can either accept or reject it.

In addition to the five states that have passed living will legislation this year, 19 others have bills under consideration. Two other states, California and Oregon (which were among the first states to adopt right-to-die laws), have amended their statutes to allow the appointment of a durable power-of-attorney to be made within the body of the living will. This is a new, modern development which strengthens both the Uniform Durable Power of Attorney law and the living-will law. In effect, this allows the maker of the living will to name a proxy to make decisions for him or her in case of later incompetence or disability. In Tennessee's new durable power-of-attorney law, passed last year, a person can condition the durable power-of-attorney upon later disability and can also name a guardian or conservator in the instrument. Much greater flexibility would be achieved with the addition of a right-to-die law.

Despite the fact that Tennessee does not officially recognize living wills, many physicians agree with the underlying philosophy and will follow their patients wishes—at least under certain circumstances.

The precise wording of a living will is not important so long as the statement unequivocally expresses the intention of the maker that he or she be allowed to die if there is no reasonable chance of recovery. The statement should be signed in the presence of two witnesses who are not blood relatives or beneficiaries of the estate of the maker of the will. Then both witnesses should sign also. Copies of the document should be given to one's doctor, lawyer, minister, and close relatives. A living will, properly signed and witnessed, and executed while one is healthy and thinking clearly, could prove invaluable in case of later disagreement among family members and physicians.

The question often comes up: Will insurance benefits be affected by a living will? The answer is no. Although there has been no court test of a living will, it is inconceivable that the document could be construed as an intention to commit suicide. The execution of a living will should have no effect on insurance.

Some senior citizens are also concerned about whether it might be against Christian principles to allow a person to die. Not so, according to most authorities. Pope John Paul II has come out in favor of the living will concept. So have the United Methodist Church, the Church of Christ, the Quakers, and the Central Conference of American Rabbis. Also the American Medical Association.

TLC thanks Donna McCrea, Legal Services Developer for the State of Wyoming, Nancy Nakayma, Institute of Public Law, University of New Mexico's School of Law and the Society for the Right To Die for material used in this article.

MEDICARE ADVOCACY

Each month hundreds of Medicaid-eligible nursing home patients are unjustifiably denied MEDICARE benefits in Skilled Nursing Facilities (SNF). Since Medicaid is a joint federal-state program, while MEDICARE is wholly federal, these denials of MEDICARE impact tremendously on the state, because most of the patients are forced to go on Medicaid. This costs the State of Tennessee millions of dollars a year.

All states have this problem, but a few are fighting back—developing advocacy programs that help elderly people appeal the unjust denials of MEDICARE. In Connecticut, the state Medicaid agency funds a special project called LAMP (Legal Assistance to MEDICARE Patients) to prosecute MEDICARE appeals for Medicaid-eligible seniors. Since most of the recovered benefits go to the state, LAMP has proven to be an excellent investment. For every dollar invested in LAMP, the state has received a return of almost $2.

This spring the Tennessee Commission on Aging sponsored a workshop on MEDICARE advocacy for its Legal Care and ombudsman programs. Conducting the training were Judith and Charles Hulin, co-founders and co-directors of LAMP. This article was stimulated by that workshop. The facts and figures are based on the Connecticut experience, which is well-documented. According to the Hulins, a similar need for such advocacy exist in all states.

Why are virtually all MEDICARE-eligible applicants for SNF turned down for MEDICARE coverage? Mainly because of the operation of a technical device called "waiver of liability," the Hulins explained, which is employed in such a way as to exclude most eligible persons.

First of all, the nursing home has the responsibility to make the initial decision as to medical eligibility for MEDICARE. This decision is then reviewed by the state fiscal intermediary (which, in Tennessee, is Blue Cross-Blue Shield). And there is the key to the matter.

If a nursing home decides a person is eligible, and the intermediary disagrees, the nursing home is not usually reimbursed for the services it has already delivered. The nursing home is punished for issuing a grant of coverage that has been overruled.

ABOUT TLC

Tennessee Legal Care is published quarterly by Tennessee Commission on Aging with funds provided by Title IV-C, Older Americans Act. The newsletter's purpose: to improve advocacy efforts in behalf of older Tennesseans by better communication and coordination among Title III-funded senior citizens law projects, Legal Services Corporation programs, the private bar, and various social programs concerned with the rights and well-being of older Tennesseans. TLC is sent free to persons employed in the Tennessee aging network. Subscription price for others: $4 per year.

Tennessee Commission on Aging
Emily Wiseman, Executive Director

Ken Floyd, Director, Advocacy Assistance

Bill Stephens, Director of Legal Care Projects, TLC Editor

But if, on the other hand, the nursing home *denies* coverage, it is seldom, if ever, overruled; and in any case it has incurred no financial liability. Nursing homes, quite naturally, soon learn to play it safe. They deny MEDICARE coverage and collect "private pay" rates until the patient uses up his or her resources and applies for *Medicaid*. Then, of course, the state has to pay . . . to the tune of big bucks.

Only a small fraction of patients who are denied MEDICARE ask for reconsideraiton, and even fewer request a hearing. In Connecticut, before LAMP, only 225 of 74,815 denials, or three-tenths of one percent, appealed to a hearing. But after LAMP was established to handle such appeals, a reversal rate of 85-90% was attained!

As originally set up, LAMP was funded primarily by the Legal Services Corporation with a supplemental grant of $100,000 from Title XX. After LAMP's demonstrated success over several years, a contract was worked out with the State, under which a special effort would be made to contact, and represent where practical, all Medicaid-eligible persons who had been denied MEDICARE coverage in SNF.

Each time a Medicaid-eligible patient is denied MEDICARE by a SNF, the Department of Income Maintenance (which handles Medicaid in Connecticut) sends a copy of the denial notice to LAMP, which offers to represent those persons on appeal. The State pays LAMP for each reconsideration request and each hearing conducted. Presently about 60 requests for reconsideration are made each month and about 30 hearings are held.

After two years of operation under the contract, it has become clear that the State is receiving a return of $1.95 for each dollar invested. About 85% of the denials of MEDICARE coverage are being reversed either at reconsideration or by the administrative law judge, resulting in an average recovery of 52.7 days of MEDICARE coverage, or $1,288, most of which goes to the State.

For further information about MEDICARE advocacy and the Connecticut experience, write to the Hulins at LAMP, P.O. Box 258, Willimantic CT 06266, or call them at 203/423-2556.

New Physician Assignment List

The new 1984 Physician/Supplier Assignment Rate List (PARL) was published on July 1 by Equitable Life Assurance Society, the Medicare Part B carrier for Tennessee. Issued in a compact 8½ x 11 size, it is much easier to use and to copy than the bulky initial version. Lists of physicians and suppliers, and their batting averages last year in accepting Medicare assignments, are easier to access and to understand.

Most important of all, the new PARL is readily available to all interested persons. Copies have been sent to Area Agencies on Aging, and to all Legal Care and ombudsman programs. A free copy will be sent on request to all consumer and public interest groups. Individuals can also obtain copies, but will have to purchase them at the cost of printing.

Requests for PARL should be made in writing to: Equitable Life Assurance Society, P.O. Box 1465, Nashville, TN 37202.

New Social Security Agency?

The Congressional Panel on Social Security Organization is expected to recommend this summer that the Social Security Administration (SSA) be made an independent agency safe from political pressures. According to Edward Roybal, Chairman of the House Select Committee on Aging, "The disaster that befell hundreds of thousands of terminated disability beneficiaries could certainly have been avoided if SSA had been more concerned with its historical mission and less worried about short-term budget savings.

"The egregious errors of the disability process have been so severe," charged Roybal, "that SSA's own employees rallied against them, and its association of administrative law judges have sued their employer, alleging they were forced to deny benefits to certifiably disabled beneficiaries."

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TO ______________________________
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TENNESSEE LEGAL CARE

TLC

Bar Training in Guardianship

Vol. 3, No. 2, Spring 1984

A seminar on guardianship and alternatives designed primarily for the private bar was presented in Cookeville in March. The program was conceived and coordinated by Lee White, attorney for Aging Services of the Upper Cumberlands, and co-sponsored by the Commission on Aging and Rural Legal Services of Tennessee. Trainers included Legal Care Attorney Nancy Preslar of Knoxville and James Mackey of Pulaski, a private attorney who is a recognized authority on guardianship and conservatorship. About 35 people, primarily lawyers, attended the seminar. Most came from Upper Cumberland counties, but some came from as far away as Chattanooga, Fayetteville, Nashville, and Jackson.

A highlight of the day was a spirited and scholarly talk by Chancellor Vernon Neal of Cookeville, who told of his experiences and concerns in the areas of limited guardianship, conservatorship, and the use of powers-of-attorney. Neal stated that some private lawyers who file a conservatorship petition think they have the right to name the guardian *ad litem*. In fact, they may even give a copy of the petition to that person before he has been appointed. *"Gibson's Suits in Chancery* sets out clearly," said the Chancellor, "that the petitioner is not a proper party to nominate a guardian *ad litem.*"

Legal Care attorneys Lee White of Cookeville and Nancy Preslar of Knoxville led guardianship training with help from bench and bar.

Chancellor Neal is particularly concerned about guardian *ad litems* not doing their job properly. All leaders of the seminar agreed that guardian *ad litems* have the duty to see the allegedly disabled person, to explain the proceedings, and to determine how his or her interests can best be served. There is a definite difference between the role of adversary counsel (mandated in the limited guardianship proceedings) and of a guardian *ad litem*. As Gibson states, a guardian *ad litem* owes a duty to the court to see that the respondent's best interests are served. The adversary counsel, on the other hand, owes a duty to the respondent and must follow his or her wishes in the matter. If the respondent is unable to communicate clearly or is confused, the attorney must oppose the petition vigorously.

James Mackey of Pulaski gave a comprehensive analysis of the conservatorship and limited guardianship statutes spiced with anecdotes from his experience as a leading proponent of guardianship at the Florida Bar. Nancy

Preslar discussed the new Durable Power of Attorney Act and stressed its versatility, emphasizing that in many cases a durable power of attorney can obviate the need for guardianship or conservatorship.

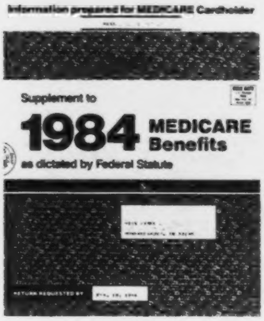
It is hoped that this successful seminar will be the first of many to acquaint the bench and bar with the pressing needs and proper remedies in this growing area of elderly law.

Insurance Company Called on Carpet

The potent influence of senior power continues to be demonstrated in Tennessee. In late winter, the TLC newspaper column, "Answers on Aging," ran two items about direct mail insurance solicitations designed to look like Social Security and Medicare notices. A great deal of response was received from throughout the state. Channel 4 television in Nashville picked up the story, as did Les Jamieson, whose 50,000-watt radio talk show has a large audience in 12 states. The U.S. Attorney's Office became interested, as did the Postal Inspector, the Federal Trade Commission, and the Consumer Protection Division of Tennessee's Department of Insurance.

The Commonwealth of Pennsylvania's Department of Insurance thanked the Commission on Aging for calling the matter to their attention and began an investigation. Charges were subsequently filed for deceptive advertising. If the insurance company is found guilty, its license could be suspended and it could be fined as much as $5,000 for each deceptive mailing, of which there were literally millions.

A "show cause" hearing was originally set for March 28 in Harrisburg. Later, on motion, the formal hearing was postponed and a "pre-trial" conference took place on March 28 and 29. After results of the formal hearing are announced, the TLC network will be informed.



Insurance mailing was designed to resemble official Medicare notice.

Information prepared for MEDICARE Cardholder

Supplement to

1984 MEDICARE Benefits

as dictated by Federal Statute

RETURN REQUESTED BY

HEALTH LAW UPDATE

Medicare and Medicaid were the first programs to go on the chopping block in March when the Senate finance committee began its attempt to cut the federal deficit. The committee approved the cutting of $10 billion from these two vital programs. "Somebody has to make the first move and I think it ought to be us," said Senator Robert Dole, committee chairman.

One of the proposed cuts would require people on Medicare to pay a $30.50 monthly physician premium by 1988, up from the $19.20 premium slated to become effective next year.

The committee's action drew fire from the American Association of Retired Persons (AARP) which said that the cuts "will result in higher costs and less access to health care for the nation's most frail population." James Hanking, AARP's assistant legislative counsel, pointed out that Medicare premiums have already increased 88% in the last four years. During the same period, physician's co-insurance has increased 100%. This, of course, does not paint a true picture of the extent of the problem. Premiums in co-insurance are only a small part of physicians' expenses older people must pay, since few physicians accept Medicare assignment.

The committee would save other funds by reducing Medicare reimbursement to clinical laboratories and to hospitals, and by reducing federal Medicaid payments to states. In addition, Medicare payments to physicians would be frozen for two years.

As anyone familiar with the operation of Medicare and Medicaid knows, the freezing of disbursements to physicians only hurts elderly people. Under that plan, even more physicians will refuse to accept assignment and continue to bill elderly people for twice the assignment rate or more. The fact that the American Medical Association, the Nashville Academy of Medicine, and other groups have recommended that doctors hold their fees at present levels is of little consequence, most observers say—a mere reaction to present Congressional pressure. Jacob Clayman, president of the National Council of Senior Citizens, called AMA's gestures "too little, too late." Clayman said that escalating medical costs are sure to result, sooner or later, in federal regulation of physicians' fees.

In an apparent effort to soften

the drastic cuts, the Senate committee recommended "incentives" for physicians to accept Medicare assignment. These incentives include:

- The publication of directories to inform senior citizens of the acceptance rates for physicians in their area;
- Expansion of toll-free phone services to provide information on physicians who accept Medicare;
- Simplification of paperwork for physicians and patients who cooperate.

New Medicare Bill

Edward Roybal, chairman of the House Select Committee on Aging, has introduced a bill to require physicians and other Part B providers to accept Medicare assignment. The "Medicare Physician Fair Payment Act of 1984" mandates that all participating providers must accept the Medicare "reasonable charge" as full reimbursement, and beneficiaries may not be charged more than the 20% co-payment.

The provision which sets this bill apart from all other plans is its requirement that all hospitals, as a condition of participation in the Medicare program, would grant staff privileges only to physicians who participate in Medicare (and therefore accept assignment).

Another Relative Responsibility Law Bites the Dust

The Idaho Attorney General has ruled that the state's relative responsibility law is inconsistent with federal Medicaid statutes and regulations and is unenforceable. Furthermore, it is not a law of general applicability, since it "imposes financial obligations only upon relatives of Medicaid recipients." Last year, the Attorney General of Tennessee, in a similar opinion, held that Tennessee's relative responsibility law is unenforceable against relatives other than spouses and parents.

Legal Care Briefs

When Physicians will not Cooperate with Medicare Patients

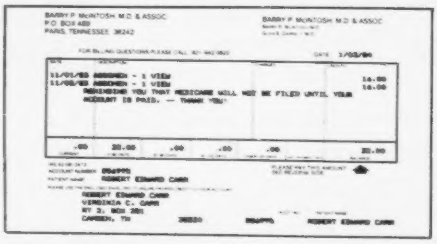
What can an elderly patient do when his physician refuses to complete Medicare forms until his bill has been paid? He can always file a complaint with the Medicare carrier, of course. But unfortunately, the carrier for Tennessee, Equitable Life Assurance Society, feels that it has no authority to enforce sanctions against physicians. Although the Medicare Carrier's Manual (Section 3307) requires all participating Medicare providers to furnish necessary medical information on claims, Equitable interprets this section to apply only to participating institutions rather than physicians or suppliers.

Section 3313 of the Carrier's Manual, on the other hand, deals specifically with the issue of physicians refusing to furnish an itemized bill (which the carrier must have in order to pay a claim in cases where the physician refuses to fill out the 1490 claim form). This section tells the carrier that if a physician or supplier refuses to furnish a bill to Medicare until they have been paid by the patient, the carrier should contact the physician or the local medical society in an attempt to resolve the problem. And as a last resort, they are told, and before disallowing the claim, they should advise the patient "to consider consulting an attorney, a legal aid society, or a consumer protection society or agency, as to whether he has an obligation under state law to pay a physician's bill on which the itemization has been refused."

Actually, it appears, as Equitable has stated to us, "that the law does not provide for a means in which to force a physician or supplier to provide such information. There is no requirement for a physician to furnish any information for a non-assigned claim."

A possible remedy may lie in the Tennessee Consumer Protection Act, however. A careful reading of the Act indicates that it is broad

Some patients are confused and outraged by doctor bills such as the one at right, and by hospital admission forms like the one below

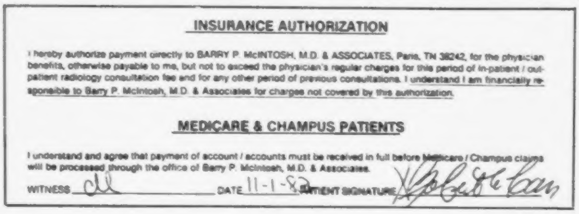


BARRY P. McINTOSH M.D. & ASSOC
P.O. BOX 480
PARIS, TENNESSEE 38242

BARRY P. McINTOSH M.D. & ASSOC

11/01/83	[illegible] - 1 VIEW	14.00
11/08/83	[illegible] - 1 VIEW	14.00
	REMINDING YOU THAT MEDICARE WILL NOT BE FILED UNTIL YOUR ACCOUNT IS PAID. -- THANK YOU!	

.00	28.00	.00	.00	.00	28.00



INSURANCE AUTHORIZATION

I hereby authorize payment directly to BARRY P. McINTOSH, M.D. & ASSOCIATES, Paris, TN 38242, for the physician benefits, otherwise payable to me, but not to exceed the physician's regular charges for this period of in-patient / out-patient radiology consultation fee and for any other period of previous consultations. I understand I am financially responsible to Barry P. McIntosh, M.D. & Associates for charges not covered by this authorization.

MEDICARE & CHAMPUS PATIENTS

I understand and agree that payment of account / accounts must be received in full before Medicare / Champus claims will be processed through the office of Barry P. McIntosh, M.D. & Associates.

WITNESS ______ DATE 11-1-83 PATIENT SIGNATURE Robert E Carr enough to apply to medical services. So, in a situation in which a physician refuses assignment and then states to the patient that a Medicare claim cannot be filed until the doctor's bill has been paid in full, the patient is not only confused, we submit, he is *deceived.* The average Medicare recipient does not realize that he can file a claim on his own.

House Vote Almost Unanimous On Disability Bill

With a resounding vote of 410 to 1, the U.S. House of Representatives passed the "Social Security Disability Benefits Reform Act of 1984." The bill is intended to stop the wholesale terminations of Social Security Disability Insurance (SSDI). Before the final vote, member after member, Republicans and Democrats alike, voiced their strong support for the bill, which was opposed by the Administration. The vote came only days after leading newspapers reported that the White House might order an 18-month moratorium on SSDI terminations. Many Congressmen who supported the new bill are opposed to a moratorium, however, on the ground that it might stall needed changes in SSDI. "Congress must not endorse this move toward lawlessness. The disabled have a right to the prompt adjudication of their claims in accordance with the law," stated Rep. J. J. Pickle, who sponsored the House bill.

Provisions of the bill include:

- persons drawing SSDI will not be terminated unless there is definite medical evidence of medical improvement.
- the Social Security Administration (SSA), in conjunction with the National Academy of Sciences, will make a study of the use of subjective evidence of pain in determining disability.
- in assessing multiple impairments, SSA must consider the combined effect of all impairments without regard to whether any single impairment, considered alone, is sufficient to meet the listings.
- face-to-face interviews will be held at the initial level of review. Before benefits can be cut off, the Disability Determination Section (DDS) must notify the recipient that he/she has a right to be interviewed before a termination decision is issued. These interviews will replace the face-to-face interviews at reconsideration, which SSA is now in the process of implementing.
- In both Title II and Title XVI termination cases, benefits may continue through the Administrative Law Judge (ALJ) level.
- SSA will follow U.S. Circuit Court of Appeals decisions throughout the applicable circuit until a different result is reached by a ruling by the Supreme Court of the U.S.

Senator Jim Sasser of Tennessee has introduced the House bill in the Senate, and progress of the bill will be closely followed by elderly advocates.

ABOUT TLC

Tennessee Legal Care is published quarterly by Tennessee Commission on Aging with funds provided by Title IV-C, Older Americans Act. The newsletter's purpose: to improve advocacy efforts in behalf of older Tennesseans by better communication and coordination among Title III-funded senior citizens law projects, Legal Services Corporation programs, the private bar, and various social programs concerned with the rights and well-being of older Tennesseans. TLC is sent free to persons employed in the Tennessee aging network. Subscription price for others: $4 per year.

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Mr. GORE. Thank you very much. We appreciate your statement.

We'd like to ask our second witness on this panel to testify. Dr. Robert D. Fox is with the Department of Medical Education and Family Practice here at East Tennessee State University.

Dr. Fox, we're honored to have you here and we invite you to proceed.

Dr. FOX. Thank you.

I'm going to make mine a little briefer than I intended to make it, because, as you said, we are running out of time.

Mr. GORE. We appreciate that.

Dr. FOX. I have submitted written testimony that documents in detail two kinds of problems, one of which has to do with the formal health care system, meaning doctors, nurses, social workers, et cetera; and that's the problem in the diagnosis of the disease that's been alluded to earlier. OK. The second problem is problems related to families and the kinds of crises that they face with the Alzheimer's disease.

In terms of misdiagnosis, it's important to note that Alzheimer's is the most recent in a series of terms referred to which began, I guess, with the label "senile." And I think Congressman Pepper referred to this earlier. "Senile" is not a medical term. It's not a term that I, certainly, will tolerate medical students using in my presence. "Senile" is a word that is entirely derogatory in nature, it's used as a social label and an indictment to a population who are, by the use of this term, identified as losing competence. And the loss of intellectual competence in this society is the loss of power. And this label has done a great deal of damage to perfectly healthy, normally functioning, older adults.

Now, we need some kind of term to refer to these problems that we're talking about, and so the other one that's used is "obstructive"—I'm sorry, that's a Freudian slip, I think—"organic brain syndrome." Organic brain syndrome is another medical term that refers to a whole group of problems, and that has its appropriate medical use. And then there is "Alzheimer's." "Alzheimer's," technically, is defined as presenile dementia. And then there is "senile dementia of the Alzheimer's type."

What I'm getting at here is that the problem that we see in society with agism as a general—and I think Robert Butler coined this phrase, as a matter of fact—agism, one of the three great "ism's" in society, pervades all of society, including the health professions. It sort of comes to light, sometimes, with the diagnosis of senile dementia of the Alzheimer's type. Because behavioral problems in old people are difficult to deal with, occasionally—and, again, I document this in the written testimony extensively—occasionally it's fairly easy to lay on the label of "Alzheimer's" or "senile dementia of the Alzheimer's type," as a way of coping with someone who has another problem, for example depression, which very closely mimics Alzheimer's.

These kinds of mistakes are often made very innocently, but are in some ways the byproduct of three kinds of forces that operate in the practice of medicine; the first of those forces being the pervasive influence of a society which disregards, ignores, or devalues its aged population. And that pervades all of society, including health care professionals. The second is that the aged patient is a difficult patient for professionals trained to cure, because aged patients typically are not easily cured. They have multiple chronic conditions, and they must be dealt with, and maintained, and assisted to improve the quality of their life; but the therapeutic goal is quite different than that of a cure in many cases, Alzheimer's is no exception.

Finally, we don't prepare our professional very well to deal with old people. Our basic standards of care, as well as our educational system are oriented toward the problems in pediatrics and adult medicine, and adult medicine is not defined so as to include, as much as it should, the problems of the aged patient population.

What we need to see is more attention in research, education, on diminishing the effect of societal attitudes in the practice of health care, improving the attitudes of health care professionals toward the aged in general, and thereby reducing, we hope, some of the misdiagnosis.

Incidentally, the latest study that I have on accuracy of diagnosis of Alzheimer's was conducted with people immediately out of medical school, in their first year of postgraduate training, and they had an accuracy rate of only 41 percent in the diagnosis. The implication here is that 59 percent of the patients in this particular study who were diagnosed as having Alzheimer's did not have it, and yet would have been treated as if they did; which means no attempt at recovery—a fairly serious problem, especially if they are depressed. They're likely to get even more depressed and become even more difficult to deal with.

The second kind of problem that relates to societal attitudes toward the aging is the attitude that we have that the formal health care system is really the way health care is delivered in the United States; and that is just not true, particularly with Alzheimer's and the aged patient. What we find is that: the informal health care, namely the family, is the principal deliverer of health care; that that system occupies and performs most health care functions; that if, in fact, we were to dump all of that health care responsibilities on the formal system, it would be completely overtaxed and unable to respond.

Our health care in the United States is dependent not only upon highly qualified professionals in health fields, but also upon families who are competent to deal with the health problems of their members. This is where we have a major problem developing in the future. Because if you think about the largest growing segment of society being the over-85 age group—which has already been noted by Congressman Pepper—who are taking care? Mostly children or spouses. That means those who are on the threshold, who are in early old age, or those who are already old are caring for those who are old and needing care, which means those with the least energies, or diminishing energies, to perform that important function, are being burdened by that function. And that's not a very optimistic picture, because it's only going to get more and more intense as we get more and more old people and as we live longer and longer.

In addition, these people are sandwiched between not only the obligations to their parents who they must care for, but as you've seen in the earlier panel, they are also maintaining their obligations to their own children, to their own families, to their own role in society. And that's why they've been referred to as "the sandwich generation" trapped between two sets of obligations, each competing for their attention and for their energy.

Primarily, these are the women of America and not the men. Basically, women have adopted responsibility for health care, for as long as we've had a society, more than men have; and yet, we're facing a future where it's not likely that that can continue to occur in the same way. We're facing a future where women have their own careers, where they have their own need to support families, where they have their own problems to face and where they're going to be less able than ever to assume care for aging parents or for spouses in difficulty.

So, in 20 years you can see a serious crisis that's sort of embodied in Alzheimer's disease already, the crisis where the demands of care are so large and the resources in the informal system to apply to that care are diminishing constantly.

The problems for the Alzheimer's families revolve around conflicting emotions, primarily. First, just dealing with an Alzheimer's victim creates a generalized fear of aging, because we still have a tendency to think that this is a problem that's going to hit everybody and that it's a problem that they may have because their family has it. That fear of aging comes directly from the contact with the person who is, in fact, having a bad aging experience. There are also conflicting emotions based upon the love that they have for the family member, the obligations that they feel and should feel, a resentment at the loss of their freedom that they should have gained when their children left home and they had the opportunity to act as a couple but are suddenly burdened with a family member, who they didn't anticipate but who they feel the love and obligation to care for. They have guilt, then, because they resent; and, so, the resentment leads to guilt and the guilt leads to, eventually, depression and loneliness; and depression and loneliness diminish their capacity to care as well as the quality of their lives. So, we have not just the bomb of Alzheimer's in the life of the victim, but we have the shock waves from the bomb that affect the lives of all that surround them.

Finances have already been alluded to adequately, I think—serious financial stress on these families.

Family disruption often follows. We don't know how many cases of elder abuse result from families who simply have lost control temporarily and have gone too far in their frustration, in their guilt, in their resentment, their anxiety, and their anger.

And premature institutionalization. Now, institutionalization of an Alzheimer's victim is appropriate at certain places. But to put them away because the informal health care system does not have the adequate support to care for the victim properly, because they've been stretched to the point of breakage, is premature institutionalization. The victim is almost always better off at home, if that could be maintained, until it is necessary.

So, the consequences of these stresses for the system are very extreme and very real. As I said, the future looks even more bleak than the present, without action: more old than ever, we have little research or understanding about this problem, about its impact, about what to do about it, and certainly health care professionals can be of only modest benefit to this system.

Finally, as I mentioned before, women may not be available, or willing, or able to continue to carry their primary role in the care for the victims. What we need is more research. We need innovative programs now, without research behind them even, because now we see the people suffer and now they need the relief.

And finally, we need a real definition of this whole problem that includes the legislation, so that this financial burden, one thing we can obviously and immediately do something about, can be relieved.

Thank you.

[Applause.]

[The prepared statement of Dr. Fox follows:]

Congressman Albert Gore Jr.'s Subcommittee Hearing on Alzheimer's Disease
August 30, 1984

Statement of Dr. Robert D. Fox

Thank you for inviting me to testify before you concerning some of the problems that surround senile dementia of the Alzheimer's type. I will present my comments in two parts. First, I want to talk about the problems which surround the diagnosis of senile dementia of the Alzheimer's type, focusing primarily upon the factors which may influence the diagnostic process. Secondly, I wish to talk about the problems senile dementia of the Alzheimer's type presents for the informal health care system, the family. It is my hope that by providing some insight into the problems of both the formal health care system, as exemplified by dilemmas associated with diagnosis, and problems of the informal health care system, as exemplified by the difficulties the disease presents to caregivers, my testimony will help establish not only some of the research needs related to senile dementia of the Alzheimer's type but also some of the priorities for action related to this tragic disease.

To be clearer about the focus of my testimony, some definitions are in order. The following are extracted verbatim from Tabor's Cyclopedic Medical Dictionary:

1. dementia - Irrecoverable deteriorative mental state, the common end result of many entities.
2. senile dementia - occurring in the aged. Symptoms: Progressive mental deterioration with loss of memory, esp. for recent events, with occasional intercurrent attacks of excitement.

3. Alzheimer's disease - Presenile dementia, which is similar to senile dementia but occurs in the 40-60-year age group. The disease has a relentless and irreversible course that may take from a few months to four or five years to go to the stage of complete helplessness.

It is from these three definitions that the term senile dementia of the Alzheimer's type is derived.

Senile dementia is severe in five percent of the population over 65 and mild to moderate in ten percent (Dans, 1979, p. 229). Approximately half the victims of senile dementia suffer from senile dementia of the Alzheimer's type. The largest growing age segment of the population is a group consisting of individuals over the age of 85. In fact, the approximately 2.5 million individuals over the age of 85 in 1980 will increase to almost 7.5 million by the year 2000 (Aging America: Trends and Projections, 1983, p. 7). Most of this age group are women, widowed and substantially poorer than the rest of the population. Senile dementia of the Alzheimer's type is more prevalent in women than men and is more prevalent among the old old than the young old. Consequently, what is now a major personal and social tragedy may eventually become a national disaster.

Senile dementia of the Alzheimer's type is difficult to diagnose. As Chart 1 indicates, (Garland, et al. 1983) senile dementia of the Alzheimer's type is mimicked by a depressive condition known as pseudo dementia. Thus, one of the major dangers related to the diagnosis of senile dementia of the Alzheimer's type is in mistaking another health problem, such as depression, for this terrible disease. When a physician misdiagnoses and thereby mislabels a patient as a victim of senile dementia of the Alzheimer's type, that patient is not subject to efforts at recovery. Labeling is accompanied by a way of relating to the patient. Because senile dementia of the Alzheimer's type is a terminal disease that involves a loss of competence, there is a tendency on the part of many to withdraw from the patient, to treat him/her like a child and to assume control over his/her affairs. Palliative measures aimed at maintenance of health dominate the patient's care. Consequently, for a patient who may be depressed or anxious but not organically impaired, the behaviors of others toward him/her may worsen his/her condition.

Several forces act to increase the potential for misdiagnosis of a patient as an Alzheimer's victim. First and foremost is a general societal attitude of negativism toward the aged. In fact, in this youth-oriented culture, fear of aging becomes an important characteristic of the emotional lives of many.

Health care professionals including physicians, nurses, social workers and therapists, are equally subject to negative attitudes toward the aged, fear of aging and dread of the aged patient (Spence, 1968). Physicians and physicians in training generally feel uncomfortable with their skills in dealing with the social problems of geriatric patients (Williams _et al._, 1982). These feelings are often coupled with inadequate training in geriatrics to create a poor understanding of aging and the aged patient. For example, Dye _et al_. (1979) found that many health professionals were unable to classify correctly disease processes versus normal processes related to aging. Miller _et al_. (1976, p. 108) found inaccurate or non-informative admission diagnosis present in 64 of 100 patients consecutively admitted to a nursing home. Pirrello (1979) found that physicians specifically were more likely than other categories of helping professionals to diagnose organically-based problems and to infer mental illness in the old (p. 548-549). Several other studies (Holtzman et al., 1978, Miller, et al., 1976, Spence, et al., 1968) have documented the inappropriateness of the attitudes of medical students, (residents and) physicians regarding the aged. These studies and others indicate a general weakness in the health care system related to addressing the problems of aged patients, but what about senile dementia specifically?

Several authors have documented the tendency of physicians and other health professionals to mislabel patients as demented (Knights and Folstein, 1977, DePaulo and Folstein, 1978, Roca et al., 1984). In a study of 57 interns' (postgraduate physicians in training) opinions regarding dementia in 380 patients, Roca et al. (1984) found that although medical interns judged that 109 of the 380 aged patients were suffering from dementia, only 57 of these patients met the criteria for dementia from DSM-III. In other words, 52 patients were labeled as demented who were not. The authors noted, "The likelihood of true dementia in the patient diagnosed as demented by interns was 41%" (p. 74). In an attempt to explain why this occurred, they noted that lower education and age of patient were often related to this misdiagnosis. Thus, patients with low social skills were more likely to be misdiagnosed as victims of senile dementia. In a classic review of literature on chronic brain disorders, Wells (1978) noted, "The conclusion is inescapable that diagnostic errors both of omission and comission are not rare, even when patients are evaluated carefully by well-trained psychologists and neurologists." (p. 2).

In addition to the problems of misdiagnosis which result from the effects of general societal attitudes toward the aged, there is also the dilemma of providing health care to a population of patients where the definition of success is different from that for younger patients. Aged patients are difficult for health professionals who are trained to cure illness. Aged patients often have multiple chronic conditions which cannot be cured, are more sensitive to medications, take many medications which may interact in unpredictable ways, are often depressed about their illnesses and anxious about their prospects. These are frustrating patients in comparison to acutely ill young adults and children who with help from health professionals may experience dramatic recoveries from single acute problems (Dans, 1979). Patients' depression and anxiety, along with health care professionals' attitudes and frustrations with definitions of success, increase the likelihood that some patients who are not victims of senile dementia of the Alzheimer's type may be labeled as such by health professionals.

A third force increasing the tendency to mislabel patients as victims of senile dementia of the Alzheimer's type may be the inadequacy of training programs in health fields. Only recently have medical schools, health schools and schools of nursing begun to incorporate significant attention to the health and illness of the aging population into their curriculum. In the past, the aged patient has been seen as just another patient, while emphasis has been placed on diseases. Recently, however, greater emphasis has been placed upon the need for many disciplines to become involved in health problems of the aged. This emphasis reflects the multiple interacting forces in the lives of the elderly. Continuing emphasis in training on the multidisciplinary nature of problems such as senile dementia of the Alzheimer's type and the concomitant need for multidisciplinary responses to those needs is an important prerequisite to success in care. Senile dementia of the Alzheimer's type is an archetype of the problems of aged patients because, like most health problems of the aged, it is manifest in social, psychological and physical domains of the patient's life.

I have focused on a specific problem related to the diagnosis of senile dementia of the Alzheimer's type by the formal health care system. Negative attitudes, inadequate training and a difficult medical situation of senile dementia of the Alzheimer's type may result in the mislabeling of a recoverable patient as incompetent and terminal. Because senile dementia of the Alzheimer's type is only totally confirmed upon autopsy, the problems of accurate diagnosis must be especially addressed in both education and research.

Given the above review of problems concerned with successful diagnosis of Alzheimer's, several recommendations seem appropriate. I recommend that more attention be paid to establishing multidisciplinary evaluation units for accurate diagnoses of aged patients specifically and multidisciplinary perspectives applied in the education of health care professionals about the aged in general. I believe it is important for education to act to prevent the influence of social attitudes and fear of aging on care for geriatric patients in general and Alzheimer's patients in particular. In addition, standards for quality health care based upon the age of the patient are needed because of the very different and complicated array of problems related to the health of the elderly. Understanding that a patient may be depressed with good reason or that the patient is anxious because two or three things are going wrong at the same time may limit the tendency of health care professionals to mislabel patients with these problems as victims of senile dementia. Finally, I believe that programs in continuing health education be devised which can be used to overcome the effect of social attitudes and fear of aging on today's health professionals. We cannot wait until better-trained health professionals dominate the health care system. The needs of those who are practicing now must be attended to immediately.

A second major set of problems related to the geriatric patient population in general and the victims of senile dementia of the Alzheimer's type in particular relates to the informal care system, the family. Brody (1981) points out several myths about health care for the elderly, including, specifically, the myth that the formal health and social services support systems provide the bulk of care received by the elderly (p. 471). She and others (Robinson *et al*. 1979, Riley and Foner 1968, Shanas, 1979) note that it is primarily the children and spouses to whom the elderly turn when they need assistance, particularly, if it's in a time of crisis. Several studies have indicated that, even in this mobile society, at least one child usually lives close enough to an adult parent to maintain daily contact with that parent (Treas, 1977, Cicerelli, 1981). In fact, most of the health care services received by the elderly in society are provided by family members, especially spouses and adult children.

This apparently bright picture is dulled by some serious problems. First, since those who need the most care are largely 75 and over, the adult children and spouses who must care for them are also either old or on the threshold of old age themselves. This means that those who are providing most of the care to those who need the most care are themselves suffering from declining energy, health and serious reduction in financial resources (Brody, 1981, Treas, 1977). Moreover, these caregivers often have children and grandchildren with all the concomitant responsibilities.

The adult children who are caring for aged parents have been referred to as a "sandwich generation", squeezed between the obligations of caring for aging parents as well as obligations to respond to the needs of their own children and grandchildren. Cicerelli (1981) notes that "Adult children (in the middle generation) tend to give more help to both the older and younger generations than they received from either (p. 4). Evidence of the magnitude of responsibilities carried by these caregivers to the ill aged is found in the fact that more old people are in bed at home than in institutions. The proportion of people between 75 and 84 who are in institutions is approximately 7.1%, while approximately 19.3% of those over 85 are in institutions. Among the non-institutionalized elderly, about eight to ten percent are as functionally impaired as those in institutions (Brody, 1981, p. 472). Of adult children who are caring for aged parents, those between the ages of 40 and 49 contribute an average of three hours weekly of help to their parents; those between 50 and 59 contribute an average of 15.6 hours of help a week, and those who were 60 years of age or older were giving approximately 23 hours help weekly to their aging parents (p. 476). For the most part, these providers of care are women.

In a recent course taught by Dr. Edith Summerlin and I to community members who were caring for aged parents, attendance was 100% female. Moreover, attendees stated that they believed that this problem was often assigned to women rather than men even when men were more closely related to the parent. According to Mindel _et al_. (1982), "Elderly people, particularly mothers, are more apt to live with daughters than with sons and to form closer and more satisfactory relationships with their daughters" (p. 485).

In regard to senile dementia, Rabins _et al_. (1982) reported that a majority of care for the demented is provided in their homes (p. 333). Gwyther _et al_. (p. 150) describes these individuals as in a "double bind situation". First, their behavior toward the demented parent serves as a model for their children if they themselves develop the disease. Second, these middle aged children are not only at increased risk of developing dementia, but also fear the responsibility of having possibly transmitted the condition to their children despite the fact that a genetic link is not well-established (p. 150). Again, senile dementia of the Alzheimer's type provides an intensive and tragic microcosm of the dilemmas faced by families as they attempt to cope with the aging spouse or parent.

The consequences of caring for the ill aged parent or spouse, in general, and the Alzheimer's victim, specifically, are dramatic. Constant contact with and care for a relative or spouse who is in intellectual decline creates a broad-based fear of aging. The impact of a relative seeing a parent or spouse in decline creates an image of one's own future which is undeniably fearful. In the "Caring for Aging Parents" seminar series presented at this University, fear and dread of aging was expressed by many of the adult children who attended, based primarily upon their experiences with their parents.

Secondly, adult children and spouses of victims of senile dementia of the Alzheimer's type often face multiple conflicting emotions concerning their situation. At a time in life when their children are leaving home and a new sense of freedom for a couple is emerging, the new responsibilities of caring for an aging parent or spouse becomes a chain, tying the spouse or the adult child to home and depriving him/her of his/her anticipated reward for 20 to 30 years of child rearing. This creates anger and resentment in the caregiver. However, at the same time, a strong sense of obligation to family is pervasive. Anger and resentment conflict with this obligation and may lead to feelings of guilt which further complicate the emotional well-being of the caregiver. In addition, obligations to other members of the family continue in the face of this crisis, creating a sense of unending responsibility which, in turn, equals an almost absolute loss of freedom. Thus, depression resulting from sadness over this loss of freedom, joins anger, resentment and guilt to form the emotional fabric of life for the caregiver.

Financially, the burden of caring for a victim of Alzheimer's is significant. Financial resources which may be applied to other purposes are diverted to the cost of care for the victim. Increased financial problems put immediate stress on the caregiver's family, further complicating the trauma of the caregiver.

The stresses and strains which result from caring for a victim of Alzheimer's very often lead to family disruptions, conflict and even crisis. In effect, the overburdened informal system of care for the victim of Alzheimer's comes to the verge of collapse. Husbands and children find themselves without the company and the assistance of wives and mothers as the Alzheimer's victim takes up most of the caregiver's time and energy. Moreover, what time there is together is time spent either focused on the problems of the victim or in a trance-like state of absolute fatigue. Our experience with participants in the "Caring for Aging Parents" seminar was that these caregivers suffer a terrible sense of being alone in their responsbility for caring for the aging relative even in the midst of their families.

The stresses on the families of the Alzheimer's victim also have an impact upon the patient. Even though the demented person may not be completely aware of all that goes on around him/her, he/she continues to be sensitive to moods and nuances, to environmental changes and to the emotional contexts within which he/she exists. Without being able to understand or explain where these feelings come from or what they relate to, patients in an already tragic situation are often co-sufferers from the tremendous burdens on the informal system of care.

Occasionally, the only resolution for this exhausting dilemma is premature institutionalization of the victim. In other words, the informal care system reaches a point at which it can no longer function and, thus, defers care to the formal system. In fact, approximately, fifty percent of all the patients in long-term care facilities are victims of senile dementia of the Alzheimer's type (Robinson, 1979, p. 587). It is unknown how many of these patients left a family in disarray burdened by guilt but stressed beyond endurance. It is also unclear how long and how well the formal health care system can serve effectively the growing number of victims of senile dementia of the Alzheimer's type.

Several other specific problems related to the informal health care system are so poorly researched that almost nothing about them is known. For example, what happens to the rural victims of senile dementia of the Alzheimer's type who have become geographically isolated from their family support system? What happens to childless widows who have almost no family support system when they develop senile dementia of the Alzheimer's type? How many cases of elder abuse are related to a highly stressed family member who simply lost control with a demented relative? We know little about the quality of services, their frequency, their effectiveness, or the value placed on these services by elderly recipients (Cicerelli, 1981, p. 6).

The future of the informal health care system is grim for several reasons. First, the burden of care for the ill aged, including victims of senile dementia of the Alzheimer's type, has largely fallen upon women. However, tomorrow's women will not be the same as the women of today. Younger women are more actively involved in careers, more dedicated to their work and may be less willing to give up these things to care for an aging parent or spouse. Already, as Judith Treas describes it, "Growing numbers of middle-aged women are asked to choose between nursing frail old parents or working to support themselves, their families and their own children" (p. 490). It is unclear how long the informal system embodied primarily in female relatives will have the capacity or willingness for such sacrifices.

Secondly, as noted before, the number of the very old in the population is increasing dramatically. This not only implies greater incidence of senile dementia of the Alzheimer's type, but also greater burdens on the informal system of care accompanied by more tragedies for individuals and families. Thirdly, very little research or education directed toward the informal care system is in process. I know of studies at Duke and at the Andrus Center at the University of Southern California. There may be others. These programs are valuable, but they seem insufficient in light of the magnitude of the problems faced by the families who must care for the ill aged, such as victims of senile dementia of the Alzheimer's type.

As with the first part of my testimony, I have several recommendations which, I think, are in order. First, I recommend that more research on the informal health care system be conducted. It is particularly important to understand in depth what kinds of crises these families face, how they face them and with what consequences. It is only through a careful understanding of the situation of caregivers that we can begin to develop appropriate ways and means to support them, to shore up the informal health care system and to improve the formal support system's ability to respond to the needs of both patients and caregivers.

Second, active experimentation and innovation in the development of supporting systems for families must proceed even without an adequate research base. The problem is already at crises proportions and promises to worsen with time. Alzheimer's is a disease that affects not only individuals but also families and societies. Any approach toward coping with this disease must be directed not only toward discovering its etiology and its cure, but also toward enhancing the capabilities of the formal and informal health care systems as they attempt to deal with it. Among the ways this may be done is support for specific activities designed to educate and enhance the abilities of the informal health care system.

Third, we must recognize a tremendous financial burden that senile dementia of the Alzheimer's type places upon families. A way must be developed whereby this burden is shared more equitably, not as an act of charity, but as an act of compassion and caring on the part of the many for the few.

I have attempted to provide information from contemporary literature on health care and aging in general and senile dementia of the Alzheimer's type in particular about problems related to the formal and informal health care systems. It was not my assumption, nor do I wish it to be yours, that this represents a comprehensive look. Rather, I have chosen to be specific about two particular kinds of problems in the hopes that by doing so, I may show you how complicated and difficult the entire situation is. The problems that I have attempted to illuminate are only two of many related to senile dementia of the Alzheimer's type. Nevertheless, they illustrate the complexity of interacting factors and consequences related to this condition. I believe that future gains lie with multifaceted, diverse research of a multidisciplinary nature directed at this and other problems associated with aging. We have anticipated this direction in the development of our proposal for a Center for Geriatrics and Gerontology at this University. Under the rubrics of the Center, we already have faculty who are developing research proposals such as the following:

1. Dr. William Wood of Psychiatry and I are planning to study fear of aging in the aged population and in health professionals.
2. Dr. Mary Swaykus of Family Practice and Ms. Rosemary Brown of Nursing are investigating the factors which lead to the labeling of nursing home patients as demented.
3. Dr. Edith Summerlin and I are planning to study the predictable crises associated with caring for aged parents.
4. Dr. Robert Lindeman of Family Practice plans to study the therapeutic effects of laughter and play on nursing home residents.
5. James Curtis of the Medical Library plans to examine types of learning activities of older adults related to health problems.
6. Dr. Steve Giles and I are working with a team from the V.A. domiciliary to establish a data base for a longitudinal study of these residents.

These activities are a sample of our future directions. We are on the edge of establishing a major thrust in the area of geriatrics and gerontology, but we must compete for support for these research activities with such prestigious and well-established centers as Andrus at USC and Duke University in Durham. Whether funding sources will invest in a new center like ours is yet to be determined. In any case, the successful development and support of multidisciplinary problem-oriented approaches to research and education in health and aging is essential if we are to proceed toward the next century with any sense of confidence about our individual futures and the futures of our society.

I thank you for the opportunity to speak about some of the issues related to health care and senile dementia of the Alzheimer's type.

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Chart 1

TABLE 8. Depression versus dementia

	Suggests depression	Suggests dementia
1.	Subjective exceeds objective signs	Converse
2.	Depression persistent	Shallow, labile affect
3.	Complains about self; exaggerates failures	Denies; or complains about others, environment
4.	Exaggerates or overreacts to memory failure	Unaware or confabulates to cover memory failure
5.	Insecure with others	Demanding of others
6.	Delusions are congruent with mood	Delusions are shallow, even ridiculous
7.	Relatively sudden onset Rapid progress	Insidious onset (unless follows a stroke) Gradual progress (unless stepwise in multiinfarct type)
8.	Less than 6 months	More than 1 year
9.	Depression precedes dementia	Converse
	Objective signs improve with depression	Temporary improvement at best
10.	May be best at night	May be worst at night
11.	Previous history of depression/mania	No depression or mania in previous history
	Previous response to antidepressives good	If responsive to antidepressants then cognitive symptoms reappeared soon
12.	Residual state normal	Residual state impaired
13.	Family history of depression/mania	Family history of dementia
14.	Relatives concerned about patient's memory	Relatives concerned about patient's disability
15.	Can find way around environment and knows where to seek help	Gets lost in familiar surroundings. Appeals to all and sundry for help

Excerpted from "Differentiating Dementia from Nondementing Conditions," in Mayeux, R. and W.G. Rosen, The Dementias, Raven Press, New York, 1983, pp. 14-15, by Barry Garland and John Toner.

Biography of Robert David Fox

I was born 36 years ago in Knoxville, Tennessee, where I lived until serving in the Military from 1966 to 1970. During this period of time, I served as an Air Traffic Controller in the United States Air Force, including one year of service in Southeast Asia. Upon leaving the air force, I returned to Knoxville where I studied for my Bachelor's and Master's Degree and served on the administrative staff of the University of Tennessee. From there, I joined the staff of the University of North Carolina-Greensboro for two years and later completed my Doctorate at North Carolina State University in Raleigh.

In 1979, I joined the faculty of the College of Medicine at East Tennessee State University as an Assistant Professor. I have since been promoted to Associate Professor of Medical Education and Family Practice.

I have authored two chapters in books, 15 publications in academic journals, and conducted 21 presentations at academic meetings. In addition, I am the principal investigator on a $65,000 grant to study change in the lives of physicians in the United States and Canada, a project which involves the collaboration of 26 academic medical centers. Within the College of Medicine, I am the Course Director of Introduction to Clinical Medicine III. My teaching activities within the College of Medicine focus primarily upon geriatrics and gerontological education, and I teach the Sociology of Aging Course in the Graduate School of the University.

When the University receives approval for its proposed Center for Geriatrics and Gerontology, I have been asked to assume the position of Director of that Center.

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8/29/84

Mr. **Gore**. Thank you very much, Dr. Fox, for an excellent presentation. We really appreciate that.

We're going to hold our questions until the full panel has been completed.

Our third witness is Ms. Eleanor Carper from the University of Tennessee. And Ms. Carper has been studying the problems confronting primary caregivers with Alzheimer's disease.

I was pleased to meet with you 2 days ago in Memphis, and we had a very interesting experience there. And we're delighted that you could attend this hearing, Ms. Carper. If you could also keep your prepared statement shorter than you had planned, we'll put it in the record; and if you could give us the highlights of it we will be delighted to hear from you at this time.

Ms. **Carper**. Thank you, Mr. Chairman. And I will try to keep my comments brief.

We wanted to look at strategies for supporting and maintaining persons with dementia in the community, realizing that such strategy must focus on the special needs of the family as well as the patient. Therefore, we did a study, looking at 64 caregivers who had patients with Alzheimer's disease. We looked at the membership roster of Memphis ADRDA—Dot King is the founder and chairman of that group—to obtain the names of the persons, and we interviewed them.

We had a structured questionnaire which asked questions about demographics, hours spent caring for the impaired person, the types of tasks that were performed by the caregiver, and the levels of social and professional support, as well as the physical, emotional, and social functioning of the caregiver.

The mean age of those interviewed was 59 years. The range was 27 to 77; 45 or 70 percent of that group were female. The caretaker was either a spouse, 66 percent of the time, or a child, 27 percent of the time, or a sibling—which there were five siblings or other relatives. Half of the caregivers perceived their health as excellent or good; 47 of the demented persons were living at home while 17 were living in nursing homes. We found that the informal and formal supports utilized by the caregivers whose relatives were in the nursing homes differed from the support used by caregivers who cared for demented relatives at home. This may mean that routine care for patients in nursing homes is provided by nursing staff while caregivers who take care of patients at home must provide total care. Although some caregivers had enough money to hire private attendants, the majority, which was 75 percent, reported that they had just enough money to get by, and 6 percent reported inadequate financial resources.

I must interject here that our study was comprised of people from higher or mid-high social economic levels, so this study might not be representative of the entire population.

Caregivers. We found that the caregivers spent a great amount of time actually physically caring for the impaired person. Caregivers who provided care at home spent an average of 82 hours per week, a range of 6 to 168 hours, while the caregivers with the relatives in the nursing home spent an average of 30 hours per week with a range of 2 to 100 hours. Managing financial matters and caring for the mentally impaired person's needs were two of the most frequent tasks undertaken by caregivers, and a majority furnished other basic needs, such as cooking, shopping, house cleaning, and laundry. We found that the caregivers who managed their patients at home spent considerably more time assisting the patient with cooking, bathroom, laundry, and that kind of thing.

When caregivers were asked to compare their level of participation in social and recreational activities prior to caring for the demented relative with the level of participation after becoming the primary caregiver, the findings were dramatic. The impact of the demanding schedule in providing care was severe on the social or recreational activities of the caregiver; 48, or 75 percent, of the caregivers reported their sleep was frequently interrupted in order to care for the patient. These problems, we found, were clearly related to the amount of effort put forth by the caregiver since those who spent more than 40 hours per week providing direct care had a greater restriction on family activities, interactions with friends and guests, and relaxed evenings.

There was a similar reduction in the sexual activities of both groups of caregivers, those who had the patients at home and those who were in the nursing home. This may be true, because in most cases the impaired elderly person was a spouse.

Caregivers were given a structured list of potential patient problems and asked which problems were present in their patient. Patient problems most frequently reported by the caregivers were: depression, confusion, dependency, and urinary incontinency. Despite the fact that this was a study involving demented elderly persons, caregivers reported confusion as a problem which they found to be especially exasperating. They also reported that falls and rage reactions were particularly difficult problems.

As might be expected under these conditions, the caregivers themselves reported a variety of physical and emotional symptoms. Various somatic complaints occurred in 51 caregivers at least weekly. These included headache, dizziness, gastrointestinal distress. Some reported sleep disturbances, difficulty in getting to sleep and staying asleep.

Caregivers were asked, in an open-ended question, to simply list the two greatest causes of personal concern; 52, or 81 percent, reported the need to get away, 49 reported feeling guilty that they, at times, were impatient with the patient. Despite such feelings of guilt, hopelessness, and anger, it is interesting to note that few caregivers, less than 8 percent, reported an increase in arguments or angry outbursts with the impaired person. Although the caregivers may have been reluctant to report such details, it appears that many coped in silent despair.

Further analyses were undertaken to determine if there was an association between the number of hours spent with the impaired person and physical and emotional symptoms experienced by the caregiver. Such disorders were as common among those caregivers who spent less than 40 hours a week providing care as those who spent more than 40 hours a week. The impairment in caregiver social and recreational activities, as mentioned previously, was significantly associated with the amount of time spent in direct care. On the other hand, the presence or absence of a private attendant in the home did not affect either the prevalence of physical and emotional symptoms or the extent of impairment of social or recreational activities.

In order to better characterize those caregivers, the 38 who reported feeling that their situation was hopeless, it was found that factors which appeared to have the greatest impact on feelings of hopelessness were the number of hours spent in direct care and the number of family members who assisted in providing that care. This may mean that when several members assisted in caring for the demented patient, the caregiver felt less locked in and had more time to devote to personal pursuits.

Caregivers were asked questions about the possibility of placing the mentally impaired person in an institution; 64 percent felt that nursing home care was of only fair or poor quality; and 40 caregivers had predominantly negative feelings about institutionalization. All caregivers had seriously considered this option for the future and had discussed this option at least once with a physician. On the other hand 49 had never discussed this option with the impaired person, and of the 15 who had, reported doing so only once.

Caregivers tended not to view their physicians as helpful in their struggle to manage their problems. When rating physician explanation of the disease process, 75 percent reported the explanation as poor or nonexistent while 15 percent rated the explanation as—I'm sorry, 23 percent rated the explanation as good. When asked about their adherence to physicians' advice, 75 percent reported their physician had never given them any advice about managing the demented patient.

What we've said here, so far, is that beyond physical impairment, the mental impairment of elders has considerable potential for the creation of burden and impact on caregivers and families. Over half of the group surveyed spent greater than 80 hours per week in direct care, providing a variety of basic services. It appears that the caregiver and impaired persons, in effect, become isolated from significant social contact.

In open-ended discussions between interviewers and caregivers, it appeared that many caregivers may be suffering from depression, most likely due to the added stresses of caring for the demented relative. We found that some of the relatives put the needs of the patients first, often disrupting their own lifestyle. The caregiver's commitment to accommodate the perceived needs of the patient can be especially stressful, particularly when the caregiver's own needs are being unmet.

Mace and Rabins have reported that many of the problems that mentally impaired persons have are amenable to physician's advice or intervention. Our study supports this contention since depression, falls, and incontinence were frequently reported and they can be treated. Many of the caregiver's problems can be improved through counseling about exercise, diet, behavioral modification, and support therapy.

Despite the fact that most caregivers sought help from physicians, they overwhelmingly reported that physician's advice was not useful or was inadequate. Additional conversations with the caregivers revealed that many were dismayed by their physician's focus on elaborate diagnostic strategies, while often devoting minimal effort to counseling, which is similar to the findings by others.

Some of the caregivers interviewed related that physicians tended to withdraw from the family once a diagnosis was made, leaving them virtually without professional assistance in the proper management of the demented patient. Since our sample size was small, and subjects were drawn from a select group, results may not be representative of the population. These issues need to be studied in other settings.

From this study we conclude that a major initiative is needed to educate and change the attitudes of the medical profession, not only the doctors but the nurses, social workers, therapists—all of those who come in contact with a patient, concerning the care of Alzheimer's patients and their families.

Some of the short-term recommendations that we felt are important are: education and awareness of Alzheimer's patient; a communication to caregivers about management of the patient by the medical profession; publicizing, networking, community resources and support agencies, letting them know what's out there for their help; the establishment of additional support groups for families, so they will have somewhere to go to just exchange ideas; providing respite for caregivers, relieving the burden of caretaking.

Some of the long-term efforts that can be done are: beefing up research efforts, searching for cause and cure; increasing gerontology and geriatrics in the medical school curricula; fund raising to establish permanent facilities for day-care centers and paid staff; allocation by the Federal Government for additional dollars for grants and ways to assist, providing outpatient care, maybe day hospitals for patients; some ways—identifying some ways—to relieve families from severe financial drains, maybe through low interest loans and that kind of thing.

Thank you very much.

[The prepared statement of Ms. Carper follows:]

Concerns of Caretakers Managing Demented Persons

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ABSTRACT

A survey was conducted of sixty-four caretakers caring for elderly relatives with senile dementia. Caretakers usually spent large portions of time caring for demented relatives, to the detriment of social, recreational and psychological function. Little support was obtained from other family members or formal social support systems, and seventy-five percent of caretakers reported receiving no advice from physicians. Professional educational programs should address specific caretaker needs in terms of advice about long-term management of demented persons.

Burden, Problems, Needs, Support

CARETAKER CONCERNS

Introduction

In recent years, concern and interest in the causation and management of senile dementia have increased (Katzman, 1976; Brocklehurst, 1977; Mace & Rabins, 1981). Strategies, such as home health care and day hospitals, are being proposed as alternatives to institutionalization for the demented person. However, studies have shown that most of the care needs for frail or mentally impaired elderly persons are still met by their immediate families (Bergmann et al., 1978; Rabins et al., 1982). Caring for frail, homebound elderly people often requires many changes and adjustments in the lifestyle of the primary caretaker (Cantor, 1983). In many cases, caretakers are likely to cut back or give up opportunities for personal pleasure, socialization with friends, vacationing and other leisure pastimes or pursuits. Therefore, any strategy for supporting and maintaining demented persons in the community must focus on the special needs of the family as well as the patient. To date, only a few studies have been conducted which attempt to define the particular types of problems caretakers have in caring for mentally impaired elderly persons at home (Bergmann et al., 1978; Rabins et al., 1982; Cantor, 1983). For this reason, we conducted a survey of the problems and needs of a group of caretakers caring for demented persons in an to attempt to determine the degree of burden and types of problems encountered in such a situation.

Methods

The membership roster of the Memphis Alzheimer's Disease and Related Diseases Association was used to obtain the names of persons caring for demented relatives or friends. All seventy persons listed on the roster in July 1982 were contacted and sixty-four agreed to be interviewed. In all cases, the interviews were conducted with the primary caretaker, defined as the person who provided the greatest number of hours per week of care for the mentally impaired person. A structured questionnaire was administered by two trained interviewers (EC and HC). Standard questions were asked about demographics, hours spent caring for the impaired person, types of tasks performed, levels of social and professional support, and questions about physical, emotional and social functioning of the caretaker.

Results

A total of 64 persons agreed to be interviewed. The mean age of the interviewees was 59 years (range 27-77) and 45 (70%) were female. The caretaker was either a spouse (42 or 66%), child (17 or 27%), sibling (4 or 6%), or other relative (1 or 2%). Half of the caretakers (32 or 50%) perceived their health as good or excellent, while the remainder reported their health as fair or poor. Eighty-four percent of caretakers had not been hospitalized in the previous three months, while 73% had seen a physician once or not at all in the same time period. In most situations, the impaired person and spouse were the only persons at home since only 24% had other dependents living with them. Forty-seven (73%) of demented persons were living at home while 17 (27%) were living in nursing homes.

The amount of support available to the caretaker is questionable. Although 41 (64%) reported some help from other family members, 32 (50%) had help less often than once a week. As seen in Table 1, a variety of formal support was used. Forty-three, or 67%, had taken the mentally impaired person to either a psychiatrist or a mental health clinic. Private attendants were used by 24 (38%) of the caretakers. This support method was utilized more often than home health or social agencies. Although some caretakers had enough money to hire private attendants, the majority (75%) reported they had just enough money to get by while 4 (6%) reported inadequate financial resources.

Caretakers spent a great amount of time actually physically caring for the impaired person, with over half spending more than 80 hours a week in direct care (Table 2). Managing financial matters and caring for the mentally impaired person's bathroom needs were the two most frequent tasks undertaken by caretakers, but a majority furnished many basic daily functions for the impaired person (such as cooking, 67%; shopping, 48%; housecleaning, 53%; laundry, 53%).

The impact of this demanding care schedule was devastating on the social or recreational activities of the caretaker (Table 3). Forty-five (70%) of the caretakers reported infrequent trips and most reported a decline in sexual activity, interacting with friends or guests, or having a relaxing evening at home. Forty-eight (75%) reported they never slept late in the morning. These problems were clearly related to the amount of effort put forth by the caretaker since those who spent more than 40 hours a week providing direct care had a significantly greater impairment of sexual activity ($p<.05$), family activities ($p<.001$), interactions with friends and guests ($p<.001$), and relaxed evenings ($p<.001$).

Caretakers were given a structured list of potential patient problems and asked which problems were present and how severe they thought the problem was, if present. As can be seen in Table 4, the rank-ordering of frequent and severe problems differs somewhat, but depression on the part of the impaired person, excessive dependency of the impaired person on the caretaker, and rage reactions were both frequent and severe. Although the majority of demented patients present with confusion, it is evident that 56% of caretakers questioned found this condition especially exasperating. Falls were seen as a frequent but less severe problem, while urinary incontinence was seen as a severe problem.

As might be expected under these conditions, the caretakers themselves reported a variety of physical and emotional symptoms. Various somatic complaints occurred (such as headache, dizziness, dyspnea and gastrointestinal distress) in 51 (80%) caretakers at least weekly. Also, 28 (43%) reported sleep disturbances at least weekly (difficulty getting to sleep or staying asleep), 24 (38%) reported frequent feelings of anxiety, and 22 (35%) reported a change in appetite (either eating too much or too little). Caretakers were asked, in an open-ended question, to simply list the two greatest causes of personal concern. Fifty-two (81%) reported the need to get away, 49 (77%) reported feeling guilty that they, at times, were impatient with the impaired person, and 47 (73%) reported feeling hopeless. Despite such feelings of guilt, hopelessness, and some anger, it is interesting to note that few caretakers reported an increase in arguments with the impaired person (5 or 8%) or angry outbursts on their own part (4 or 6%). Although the caretakers may have been reluctant to report such details, it appears that many coped in silent despair.

Caretakers were asked several questions about the possibility of placing the mentally impaired person in an institution. Most (41 or 64%) felt nursing home care was of only fair or poor quality. Interestingly, those caretakers who had had relatives in nursing homes in the past tended to have a more adverse view of nursing homes since 77% of these caretakers rated nursing home care as fair or poor, while only 48% of the caretakers who had never had a family member in a nursing home rated the care as fair or poor. Forty of sixty-two (65%) caretakers had predominately negative feelings about institutionalization. All caretakers had seriously considered this option for the future and all had discussed this option at least once with a physician. On the other hand, forty-nine (77%) had never discussed this option with the impaired person, and the 15 (23%) who had, reported doing so only once.

Caretakers tended not to view their physicians as helpful in their struggle to manage their problems. When rating physician explanation of the disease process, 46 (72%) rated the explanation as poor or nonexistent, while only 15 (23%) rated the explanation as good. When asked about their adherence to physicians' advice, 48 (75%) reported their physician never gave them any advice about managing the demented patient.

We undertook further analysis to determine if there was an association between a number of hours spent with the impaired person and physical and emotional symptoms experienced by the caretaker (gastrointestinal symptoms, dizziness, anxiety, sleep disturbances, etc.) Such disorders were as common among those caretakers who spent less than 40 hours a week providing care as those who spent more than 40 hours a week. The impairment in caretaker social and recreational activities, as mentioned previously was significantly associated with the amount of time spent in direct care ($p<.05$). On the other hand, the presence or absence of a private attendant in the home did not affect either the prevalence of physical and emotional symptoms or the degree of impairment of social and recreational activities. Interestingly, there was no relationship between employing a private attendant and the number of hours spent per week in direct care. Finally, to better characterize those caretakers who felt their situation was hopeless, multiple regression analysis was undertaken to determine whether the number of hours spent in direct care, family support, various patient-related problems, social activities and caretaker health were associated with feeling hopeless. The variables which were associated with feeling hopeless include spending more than 40 hours per week in direct care (p<.001), presence of urinary incontinence (p<.05), confusion on the part of the impaired person (p<.05), and caretakers feeling the impaired person was too dependent (p<.001).

Discussion

This interview survey indicates that caretakers who care for mentally impaired elderly persons spend enormous amounts of time in direct care to the detriment of social and recreational function and probably to psychological function as well. Over half of the group surveyed spent over 80 hours per week in direct care, providing a variety of basic services. Unfortunately, most of the burden tends to fall on the primary caretaker, usually the spouse, since less than half saw other family members as frequently as once a week and use of other social support systems was sporadic. Nearly all caretakers report a dramatic decline in interactions with friends, guests and family members. It appears that the caretaker and impaired person, in effect, become isolated from significant social contact. Both Grad and Rabins have linked caretaker social isolation with potential emotional problems (Grad & Sainsbury, 1963; Rabins et al., 1982).

Open-ended discussions between interviewers and caretakers revealed that many caretakers may be suffering from depression, most likely due to the added stresses of caring for a demented person. We found that some of the relatives caring for a demented patient put the needs of the patient first, often disrupting their own lifestyles. This need to accommodate, at whatever sacrifice, the perceived needs of the patient, can be especially stressful to the caretaker, particularly when the caretaker's own family's needs are not being met.

Spouses of demented individuals often sacrifice their independence in order to be at home with the patient 24 hours a day, believing that this is the right thing to do (Reifler & Wu, 1982). They discontinue most social functions and recreational pastimes to accommodate the needs of the patient.

Mace and Rabins (1981) have reported that many of the problems that mentally impaired persons have are amendable to physician advice or intervention. Our study supports this since depression, falls and incontinence were frequently reported and can be treated. In addition, many of the impaired person's problems, such as sleep disorders, bowel problems, rage reactions, conflict and guilt, can be improved by counseling about exercise, diet, behavioral modification, and support therapy.

Despite the fact that most caretakers sought help from physicians, they overwhelmingly reported that physician advice was not useful or inadequate. Additional conversations with the caretakers revealed that many were dismayed by their physician's focus on elaborate diagnostic strategies, while often devoting minimal effort to counseling. Similar findings have been reported by Barnes (Barnes, et al, 1981). Some of the caretakers interviewed related that physicians tended to withdraw from the family once a diagnosis was made, leaving them virtually without professional assistance in the proper management of the demented relative.

Our findings lead us to conclude that a major initiative is needed to educate and change the attitudes of the medical profession concerning the care of demented indiviuals and their families. Since our findings indicate that caretakers experience deleterious changes in social, recreational, and psychological function, it is crucial that these variables be addressed in periodic counseling sessions.

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Table 1. Sources of Formal Support

Psychiatry	25	(39%)
Private Attendant	24	(38%)
Mental Health Clinic	18	(28%)
Social Worker	12	(19%)
Home Health	10	(15%)

Table 2. Primary Caretaker Effort

Hours spent per week, direct care,		
greater than 80	33	(52%)
median hours	72	(0-166) range
Types of frequent assistance:		
bathroom	48	(75%)
cooking	43	(67%)
medications	35	(54%)
finances	64	(100%)

Table 3. Impact on Social Activities

Activity	Frequency		
	More or Same	Less	Never
Trips	11 (17%)	45 (70%)	8 (12%)
Friends or Guests	15 (23%)	43 (67%)	6 (9%)
Relaxing Evening	18 (28%)	41 (69%)	5 (8%)
Sexual Activity	7 (11%)	51 (80%)	5 (8%)
Sleep Late	10 (16%)	6 (9%)	48 (75%)

Table 4. Caretaking Ranking of Management Problems

Frequent		Severe	
Depression	(62%)	Dependency	(20%)
Confusion	(56%)	Urinary Incontinence	(20%)
Dependency	(54%)	Depression	(19%)
Falls	(46%)	Rage	(18%)
Rage	(44%)	Fighting	(18%)

Mr. GORE. Thank you very much for a fine statement. We appreciate that.

[Applause.]

We have only two more witnesses, and I'm going to ask Dr. Steven Berk, Chief of Medical Service at the Veterans' Administration Medical Center at Mountain Home if you would come and join this panel at the witness table—we're going to put in another chair—because we can save a little time that way, if that's possible.

Thank you very much.

Our next witness is Mrs. Mary M. Lancaster, a registered nurse with the Veterans' Administration Center at Mountain Home.

Mrs. Lancaster, we're delighted to have you here, and we invite you to proceed with your statement. If you can summarize your prepared statement, we'll put it all in the record; and we thank you for your understanding about our time pressures. Please proceed.

Mrs. LANCASTER. Thank you very much. I appreciate this opportunity to speak on behalf of the many family and professional caregivers of individuals with Alzheimer's.

I am the cofacilitator of the Alzheimer's family support group which serves upper east Tennessee and southwest Virginia. I would like to concentrate on the needs and problems of caregivers as they have been related to me by the support group members. And I think it is very important, for this testimony, to remember that the word "caregiver" refers to both family and professionals.

Caring for a person with Alzheimer's disease in any setting is physically and mentally demanding. Institutional care is no less demanding, yet there are two basic differences; the first being that there are more people to share the work load, and second, that the staff is able to leave the situation at the end of 8 hours.

Mr. GORE. I wonder if you could move that microphone around to the side, so as to get it a little bit closer to your mouth.

Mrs. LANCASTER. All right. Better?

Mr. GORE. Yes. Thank you.

Mrs. LANCASTER. This lack of in-home help may be the greatest factor which results in the institutionalization of the person. Lack of information, education and training of both family caregivers and professionals is a major problem. Caregivers need to understand the disease itself, so that they can anticipate and cope with the many and varied manifestations of the disease. They need to know what is the best way to manage certain behavior such as memory loss or urinary incontinence.

This also brings to mind the lack of adequately researched and documented methods of management. What is the best way to take care of this problem? What is the best way to prevent or delay deterioration of the disease?

This lack of education and training to adequately manage the individual is a major source of frustration for caregivers. Caregivers need to be equipped with the skills which will help to reduce those instances that cause frustration and foster feelings of helplessness. This education and training is needed not only for the primary caregivers but also for support services such as physical therapy and speech therapy.

Long-term care facilities have their own problems and needs in the delivery of care to the individual Alzheimer's. They face problems of staffing shortages, poor facility design, and lack of therapeutic programs for the Alzheimer's person. Staffing shortages may mean that the employee must go ahead and do for the patient instead of instructing them and supervising them in continuing to do for themselves, since the former is less time consuming.

Building design, such as the long, ground-level facilities, are often inappropriate to house the person who wanders and has reasoning and judgment impairment. This person is sometimes able to leave the facility and runs the risk of injury or becoming lost. The lack of appropriate therapeutic programs and outside areas in which the patient can move about freely yet safely brings with it a decrease in the self-esteem of the patient and a more institutional environment.

Because long-term care facilities face many of these problems, some must resort to medications and restraining devices to aid in the control of the behavior. In addition, many facilities must limit the number of Alzheimer's patients which they are able to care for. This, then, causes a placement problem for the family. This may be because the facilities are recognizing the time and skill that is required to care for the individual with Alzheimer's, and that they cannot adequately and or safely provide that care.

I want to emphasize at this point that long-term care is a viable and maybe the only option available to some families. Resources and research need to go not only to searching for alternate forms of care but also to improving the nursing home situation, which is a needed and appropriate level of care for many, many people.

Lack of home help is a very major problem. It's been estimated that 10 to 30 percent of the patients in nursing homes could be managed outside of the facility if the resources were available. There is also a problem of publicity and coordination of community services—a full range of respite services needed, which would include in-home help, day care, short-term institutional care, congregate living, foster care—all these types of things. All of these can provide the needed time away that replenishes the caregiver so that they can continue their caregiving.

The caregiver needs to know what is available to them and need single-point-of-entry case management. Seeking out services consumes very valuable time that the caregiver may not have.

The financial burden which these families face has been recognized. We're talking about care that requires maybe 10 to 15 years.

Finally, there is a great knowledge gap in the lay community and in the professional community. That has already been discussed. The community needs to be aware of the disease and to be sensitive to the needs of the caregivers. Social isolation is a major problem faced by caregivers. The caregivers want and need to know what is going on in research and in Government policy. The media needs to be aware that the tendency to sensationalize research findings can be detrimental to the caregiver. And most importantly, there is a nationwide need for education that Alzheimer's is not a part of the normal aging process but a disease which requires a careful diagnosis and treatment. Through this greater awareness and understanding comes the hope for better health care and quality of life for the victims and their families.

Self-help support groups assist in meeting some of these needs of the caregivers. These groups provide information, education, and a time for the needed social support and contact. The group serves as one source of respite for the caregiver, yet is also educational. The caregivers share their problems and their solutions; they teach one another. As a result of these groups, community awareness may be increased, institutionalization may be delayed, and caregiver burnout may be delayed and or avoided.

These groups care for the second victim of Alzheimer's, the caregiver. These groups provide an untapped source of information, solutions, and energy. At the present time we have two support groups here in upper east Tennessee: One meets at the Veterans' Administration and one meets in Bristol. If anyone wants further information, they can contact me.

Some of the most significant needs, and concerns, and problems faced by the caregivers have been summarized. Meeting these needs and finding solutions lie ahead. Continued support of the family support networking system and the national association is needed. A comprehensive, integrated range of community based services needs to be developed. Single-point-of-entry case management needs to be expanded. Informational and educational programs need to be provided. Research into the ways of managing the day-to-day care must be carried out. Local, State and Federal governments must recognize the special added costs and burdens of caring for the individual with Alzheimer's.

And finally, I believe a pat on the back for a job well done, and a hug to show that we care, and a visit to alleviate the isolation must be provided to the family caregiver. We must defeat this enemy before it defeats us.

Thank you.

[The prepared statement of Mrs. Lancaster follows:]

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Prepared Statement of Mary McLeod Lancaster, RN, MSN
Clinical Nurse Specialist in Gerontology
Veterans Administration Medical Center, Mountain Home, Tennessee

Thank you very much for this opportunity to speak on behalf of the many caregivers of Alzheimer's patients. I am the Clinical Nurse Specialist in Gerontology at the Veterans Administration Medical Center in Johnson City and co-facilitator of the Tri-Cities Area Alzheimer's Family Support Group. I am involved in the direct nursing care of the Alzheimer's individual in both acute and long-term care settings. In addition, I work closely with families and other caregivers of Alzheimer's victims in the community.

No one testimony or story can adequately convey the complex network of problems, needs, solutions, etc. which are a part of this "Silent Epidemic." Hopefully, this testimony along with all others will help to increase the understanding of the impact that Alzheimer's Disease has on the individual, the family, health professionals, and the health care system, and serve to stimulate some positive action which will help to bring about better health care and quality of life for the patients and their families. I would like to concentrate in this testimony on the needs of the Alzheimer's care givers. It is important to remember that 'caregiver' refers to both family members who provide home care and health professionals .

Caring for the Alzheimer's patient in any situation (home or institution) is a physically and mentally demanding job. Institutional care is no less demanding than in-home care; however, there are more people to share the workload, and at the end of the shift the staff can escape the situation. This does not apply to in-home care where there is generally only one or two people to share the workload over a twenty-four hour period. This lack of in-home help may be the greatest factor which results in the institutionalization of the person. Generally, the decision to seek institutional placement is made out of necessity, and not out of choice. I want to emphasize that institutionalization may be the only viable option for many families and that placement in a nursing home is a very appropriate alternative.

The amount of care required for the person depends largely on the stage to which the disease has progressed. Generally, nursing home placement does not occur until late stage two or stage three. The moderately impaired person may require assistance with bathing and dressing, close observation to prevent injury or wandering, activity planning to delay physical and mental deterioration, and supervision in many other areas. This type of care requires approximately 3½ hours of direct nursing care in an eight hour day. The severely impaired patient who cannot walk, turn, or feed himself, and is incontinent may require up to 4½ hours of care per eight hour day. As you can see, a significant amount of time is devoted merely to the basics of care. In addition to this basic care, the time utilized in documentation, planning, conferences, looking for misplaced clothing and inservicing must be recognized.[1]

Both family and professional caregivers lack adequate education and training to effectively manage and care for the Alzheimer's person. Many of the typical rehabilitation approaches used with other chronic conditions do not work with Alzheimer's because of the cognitive impairment involved. Short attention span and memory impairment mean that instructions must be very simple, and repeated numerous times only to be forgotten by the next day. This is a source of extreme frustration for the caregiver. Activities must be short in duration and repeated throughout the day, and more time must be allotted to complete a single task. This type of education and training is needed not only for the primary caregivers but also for support services such as physical therapy, speech, occupational therapy and recreation.

Another significant problem which is identified by caregivers is the lack of adequately researched and documented methods of management. That is, what works best in any given situation. How should disorientation be handled? Does a structured program of physical activity help to delay deterioration? Does memory retraining work with the Alzheimer's patient? Questions like these have been asked by both family and professional caregivers. The caregiver needs to be equipped with the skills necessary to decrease[2] the frequency of everyday occurrences that are a source of frustration.

Staffing shortages in many institutions often do not allow the employee the time required for rehabilitation/prevention techniques. It is often much easier and quicker to feed a patient than to take the time to instruct the patient in the use of the spoon and to remain available for encouragement. Also, because of the lack of understanding of the disease, many caregivers are unable to deal effectively or therapeutically with the mental/psychiatric manifestations of the disease.

The physical structure and layout of many nursing homes is not suitable for housing a person who suffers memory impairment, who may wander or elope and possibly become injured. Very frequently, nursing homes are long, ground-level structures with many entrances and windows which provide an easy exit for someone whose reasoning and judgement are impaired. There is also a lack of appropriately designed outside areas which allow these individuals to explore with a sense of freedom and to continue to enjoy the out-of-doors, thereby maintaining a feeling of dignity and self-worth. A therapeutic park program has been developed at the Veterans Administration in Salisbury, North Carolina which allows the individual who has memory impairment, who may wander and become lost, to move freely in and out of doors as they please, yet provides them with a safe, enjoyable environment. A large grassy area adjacent to the facility has been completely enclosed with a high fence (which has been hidden by planted shrubbery). A maze of sidewalks have been constructed for wheelchair patients and the entire area has been landscaped. There is a patient vegetable garden for use in the summer and picnic areas where patients and families can be alone.

Because of the problems with staffing, education, poor resources and building structure, many facilities that care for Alzheimer's individuals must resort to restraining devices and/or medications to aid in the control of behavior. In addition, there are complaints from families that they are unable to find a nursing home that will accept their loved one. Maybe this is because nursing homes recognize the amount of time required to care for these patients and that they cannot safely or adequately care for the patient without significantly diminishing the patient's quality of life. Resources need to be directed, not only to searching for alternate forms of care, but also to improving the nursing home situation - a needed and appropriate level of care for many, many Alzheimer's people.

The quality and quantity of care which is often expected by families and society is often not achievable because of poor staffing, poor resources, and lack of education and training. If the type of care which is expected is to be delivered, the resources to do so must be provided.

As mentioned earlier, nursing home placement is frequently the only option open to families. However, there are numerous families who could and would continue to provide in-home care of the victim if the resources to do so were available.[3] It is estimated that 10-30% of the patients in nursing homes could be managed at home if resources were available. Spouses must often quit work to care for the patient, or they must pay to have someone sit with the patient while they work. In either event, this is a real financial burden - especially when you consider that the disease may run a course of 10-15 years.

There is a definite lack of affordable community resources and coordination of services in terms of in-home support. Most senior centers have a day care program, but these are not geared towards the needs of Alzheimer's individuals. In Washington County, Tennessee, there is, to my knowledge, only one day care program which would serve the needs of the Alzheimer's patient and family. However, this program serves only 35 people per day and the family must meet an income requirement. Another daycare program, Alzheimer's Disease Day Treatment, which is one of the services available through the Wisconsin Regional Geriatric Center in Milwaukee, has been in existence since 1981. This program provides a nursing assessment of the patient for functional status and presenting problems, a recreational assessment for interests and skills, and a social work assessment for social, emotional, and financial needs. The day care is available 5 days/week and members pay according to ability up to $25 per day. In addition to having someone to care for the person, the person is involved in group activity and experiences social stimulation.[4]

The need for respite care which consists of home care, day care, and short-term institutional care is dramatic. Respite services provide the caregivers with a break from the daily routine of caregiving and affords them the time to engage in needed social and recreational activity as well as tasks such as grocery shopping. The availability of respite services is a major factor in the prevention of premature institutional placement. A model program in the state of New York is called the Natural Supports Program. The emphasis on this program is to help the individual and the caregiver stay together through supplementation of family care, not the substitution of care.[5] Some of the nursing homes in the Tri-Cities Area are offering short-term respite care in which the family can admit their loved one to the nursing home for up to two weeks. Congregate-living homes for Alzheimer's persons is yet another alternate form of care which is available in Michigan and Florida.[6]

Not only is there a lack of services, but there is a need for publicity and coordination of services. Many families either do not know what is available or they go from agency to agency seeking out the needed services. This can consume very valuable time and be extremely frustrating and exhausting for the caregiver. Access to clients for the Comprehensive Elderly Service System (ACCESS) is a model program in eight upper East Tennessee counties which is funded through the Robert Wood Johnson Foundation. ACCESS serves the elderly citizen/family in seeking out and coordinating needed community resources. The program is an information and referral system which works closely with and through other area agencies. ACCESS provides an in-home needs assessment, makes referrals to agencies, and provides follow-up service. This type of program is invaluable to the elderly citizen. Another form of community service which has recently gotten underway in St. Petersburg, Florida is the establishment of the Alzheimer's Foundation which is a clearinghouse for information on Alzheimer's Disease. It is hoped that this foundation will rapidly expand to become a nationally recognized training facility and research center for health care professionals.[7]

Financial assistance is a great need of family caregivers - one which has received a lot of attention. The cost of in-home care and nursing home care is staggering. At the present time, the tremendous cost of long-term care for Alzheimer's individuals is borne almost entirely by families, and the vast majority of the patients and spouses are elderly, making this responsibility even more burdensome.[8] Medicaid requires, in most instances, that assets be "spent down" to an absolute minimum before the person becomes eligible to receive benefits. This frequently leaves the family with little, if any, income on which to survive. Medicare does not cover the type of care required by most of the patients. In general, private insurance companies do not reimburse for in-home or nursing home care.

Added to these financial burdens is the lack of understanding of the nature of the disease by disability programs. I have been told by a spouse that her husband did not qualify for disability because he was not physically ill. The monies that are available for assistance are biased for long-term care. However, approximately 2/3 of the Alzheimer's persons that have families are cared for at home and another 1/3 of those who do not have families are cared for outside of institutions.[9] What is needed is an integrated long-term care program which draws on all alternate forms of care which is partially funded through grants and Federal assistance.

There is a fundamental need for understanding, awareness, and education for the caregivers. Families have called after leaving the doctor's office desperately searching for information. "The doctor said that my husband has Alzheimer's and that there is nothing that can be done." They need education in all areas of the disease and care from diagnosis, to how to handle incontinence, to autopsy, to legal and financial counseling. The community at large needs to be aware and understand the disease and become sensitive to the needs of the caregivers. Social isolation is a major problem for caregivers. Education to the need for accurate screening and diagnosis and follow-up is extremely important. Insurance companies, policy makers, local/state/federal representatives need to understand the tremendous impact that this disease has on the nation. Caregivers need to know what is going on in the way of research, public policy, and government programs so that they can maintain a ray of hope. They need to be cautioned that the media tends to sensationalize research findings and that there are people who will take advantage of their desperate situation. And most importantly, there is a nationwide need for education that Alzheimer's is not a part of the normal aging process, but a disease which requires a careful diagnosis and treatment. Through greater awareness and understanding comes the hope to change the public attitude toward Alzheimer's Disease.

How can many of these needs be met? Through self-help Family Support Groups. The major goals of support groups are mutual aid/support, education and advocacy. Education is geared toward family members, professionals, and the community. In addition to education is the provision of mutual aid and support. Members discover that they are not alone, that there are people who are willing to help and who can be called upon for help and/or advice. The National ADRDA began as Family Support Groups. Individuals experiencing similar situations, getting together to talk and share and socialize. The National has grown to over 103 chapters and affiliates and several hundred support groups nationwide.

One such group exists in Johnson City. Originally started in 1982 with a mailing list of twenty, the group has expanded to a mailing list of 85+ and another group has started in Bristol. As a result of this type of group, community awareness has increased, institutionalization has been delayed, and caregiver burnout is delayed and/or avoided. The support group is to care for the second victim of Alzheimer's - the caregiver. Long-term care services must meet the needs of the caregiver as well as the individual if they are to be successful. They learn to cry, to laugh, to be angry and deal with it, and how to overcome guilt. In a time when health care costs are skyrocketing, it is nice to know that people are still willing to band together to help themselves. In some way, this gives the caregiver the sense of fighting back.

Research is being planned to examine the factors which influence the success or failure of support groups. Research carried out with 500 caregivers in North Carolina demonstrated that family caregivers experience more stress, take more psychotropic medications, are less satisfied with the quality of leisure time and participate in fewer social activities than the general population. The findings show that caregivers report 3X as many stress symptoms as the general population. The study also demonstrated that support[10] groups ensured that caregivers were aware of community resources.

In closing, some of the most significant needs of the caregivers have been identified. Caregivers need information, and education, financial assistance, in-home, day care, and short-term institutional respite care, the support of health professionals and society, availability and coordination of community services, and hope. How can these needs be met? Continued support of the National ADRDA and family support networking system for the dissemination of information and to function as the major advisory source for government agencies and Congressional activity. Provision of family and professional educational forums to alleviate ignorance and misconceptions. Provision of a full range of integrated community services to give the caregiver the needed respite and to enable the family and patient to remain together as long as possible; avoiding premature institutionalization and caregiver burnout. Recognition by the Federal tax structure of the special added costs of caring for an Alzheimer's person. Development and revision of private, state, and Federal insurance programs to include both in-home and institutional care of the Alzheimer's person. Recognition of Alzheimer's Disease as a total and permanent disability. Continued support of research efforts and coordination of such efforts. And most of all, a pat on the back for a job well done, a hug to show that we care, a visit to help alleviate the isolation, and just plain being there. The needs of the caregivers as well as the patients must be met before they succumb. Alzheimer's Disease is destroying the minds and bodies of our friends and relatives. It is taking a toll on the caregivers, breaking them physically, mentally, emotionally, and financially.

We must take up arms against this enemy before it destroys each and every one of us.

Thank you.

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Mr. **Gore**. Thank you. A very eloquent and helpful statement. All of them have been.

Before going to you, Dr. Berk—let me just ask you right away, Mrs. Lancaster: If someone here wanted to contact your support group, how would they go about it, just contact you, Mrs. Mary M. Lancaster at the Veterans' Administration Center——

Mrs. **Lancaster**. Yes, sir.

Mr. **Gore** [continuing]. Mountain Home, TN? Just write you a letter or call you?

Mrs. **Lancaster**. They can write me or call me, or they can call me at home.

Mr. **Gore**. All right.

Now, the second support group meeting in Bristol, is that the one that Mrs. Beatrice Abernathy is organizing?

Mrs. **Lancaster**. No, Mrs.——

Mrs. **Abernathy**. No. I'm with Mary.

Mr. **Gore**. You're with Mrs. Lancaster's group. All right, fine.

What is the second one?

Mrs. **Lancaster**. Jane Shore at the Bristol Nursing Home is the facilitator of that group, and she can be contacted at the Bristol Nursing Home.

Mr. **Gore**. Jane Shore. Do you know how you spell that?

Mrs. **Lancaster**. S-h-o-r-e.

Mr. **Gore**. S-h-o-r-e—at the Bristol Nursing Home.

OK, we have one more witness, and then we're going to have a couple of brief questions. And then we'll conclude the hearing. And don't forget that signup sheet at the end of the hearing.

Our last witness is Dr. Steven Berk, Chief of Medical Service at the Veterans' Administration Medical Center at Mountain Home.

Dr. Berk, we're delighted and honored that you could join us here today. Please forgive us for requesting that you shorten your formal statement because we've run out of time, but if you could do so we'd appreciate it. Your full text will appear in the record and we invite you to proceed.

STATEMENT OF DR. STEVEN BERK, ASSOCIATE PROFESSOR AND CHIEF, DIVISION OF INFECTIOUS DISEASE, EAST TENNESSEE STATE UNIVERSITY, QUILLEN-DISHNER COLLEGE OF MEDICINE, AND CHIEF OF MEDICINE, VETERANS' ADMINISTRATION MEDICAL CENTER, MOUNTAIN HOME, TN.

Dr. **Berk**. I'll just make some additional comments as an internist and as a clinician teacher.

I think it is important to reemphasize that dementia is a disease process. We recognize that there is some intellectual deterioration that is probably related to aging itself, yet we also know that many of the great thinkers, leaders, scientists, have been elderly people who belie the view that intellectual deterioration in the elderly is inevitable.

Dementia is an abnormal deterioration in intellectual capacity, and all elderly patients who experience mental deterioration that affects day-to-day living need medical evaluation. Before diagnosis of Alzheimer's is made, we must be sure that no reversible cause of dementia can be found. No matter how many elderly patients with mental deterioration there may be in the United States at any given time, we must guarantee that all are thoroughly evaluated for the cause of their intellectual impairment, for many studies and our own experience have shown that a percentage of older patients with dementia will have a disease for which the dementia can be reversed. These patients can be returned to a life of normalcy with proper diagnosis and therapy.

Such diseases as hypothyroidism, certain vitamin deficiencies, infections of the nervous system, pernicious anemia, brain tumors, can cause mental deterioration in the elderly but be treated effectively after diagnosis. It is therefore recommended that all patients with dementia have a complete physical examination as well as a series of laboratory tests, including blood count, thyroid function, urine analysis, chest x-ray, blood chemistries, and perhaps CAT scan and lumbar puncture.

It must also be recognized that elderly patients with depression, with hearing loss, and with some side effects of medication may also be misdiagnosed as dementia. But if no reversible cause for mental deterioration can be found, then Alzheimer's becomes a most likely diagnosis. As previously discussed, there are no definitive tests to prove this disease, although at autopsy there are classic pathologic findings in the brain. And the severity of these abnormalities by microscopic study correlate with severity of mental impairment.

Once the diagnosis of Alzheimer's is made, the goals of our health care team are to make the patient's life as comfortable and meaningful as possible, maintaining an optimistic approach while recognizing that some degree of continued mental deterioration is inevitable. In an institutionalized setting the emphasis is on a multidisciplinary approach to care, recognizing the patient is as much an individual as he was prior to his disease.

The patient in an institutional setting should be seen by social workers, internist, psychiatrist, rehabilitation therapist, and nurse specialist. The social worker must help preserve the patient's ties to his family and help the family deal with issues of guilt and rejection that occur with institutionalization. A psychiatrist can help assess the possibility of depression as a concomitant illness, and psychotropic medication may be prescribed.

The internist must come to realize that his patient is a fragile one, that the life span of a patient with Alzheimer's disease is usually diminished. One reason for the high mortality rate of the elderly patients with Alzheimer's disease is that the patient is less likely to report symptoms of serious illness. The chest pain of heart disease or the shortness of breath of pneumonia' a common causes of death in the Alzheimer's disease patient, may go unreported and unnoticed.

The Alzheimer's disease patient, then, much like the very young child, needs special attention. Those with training, and occupational, and arts therapy may help the patient adjust to his declining mental capabilities. Hobbies, interests and pastimes must adapt to these limitations. In that regard the development of programs for the patient with Alzheimer's disease must take into account regional differences. The patient who has spent his entire life in the hills of Tennessee might not do well in a support program designed for a patient from New York. Many Alzheimer's disease patients have an urge to wander. Institutional confinement is likely a more severe restriction to a patient who has lived on a farm than one previously confined to an apartment.

And last but not least among the health care team, the well-trained nurse becomes a patient's greatest friend. The quality of a day-to-day existence, where little things are all that are left, depends on a trained, compassionate nursing care team.

Ideally, an institutionalized setting should adapt to the patient's level of deteriorating function. And without going into any details, at the Mountain Home VA we have a domiciliary, a nursing home care unit, intermediate care unit, and acute care hospital, all of which have Alzheimer's disease patients and can respond to an increasing limitation that's the natural course of this disease.

I'll say very little about research into Alzheimer's disease, since several with a much better experience in this than me have already discussed research possibilities. I would like to say that we will likely be supporting and caring for patients with Alzheimer's disease for many years; and while physicians and basic scientists may be the focus of most research support, it is possible that research into methods to best provide quality of life for the Alzheimer's disease patient and family will be at least as important. Home health care, day care, and innovative institutionalized settings are attractive areas for research support. Nursing homes must be established which become centers for research and teaching about the problems of Alzheimer's disease and aging in general. At the Veterans' Administration Medical Center and ETSU College of Medicine, we hope to develop the best possible approach to the care of our dementia patients to teach and inspire our students to become physicians sensitive to the challenges of aging and to use our unique resources to contribute to the understanding of this disease. Thank you.

[The prepared statement of Dr. Berk follows:]

UNITED STATES HOUSE OF REPRESENTATIVES

COMMITTEE ON SCIENCE AND TECHNOLOGY

ALZHEIMER'S DISEASE

Steven L. Berk, M.D.
Associate Chairman
Department Internal Medicine
Quillen-Dishner College of Medicine
Chief of Medicine
Veterans Administration Medical Center
Mountain Home, Tennessee

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It has been estimated that by the year 2020 there will be 55 million Americans over the age of 65, representing 20% of the total population. Five percent of these elderly patients will have some kind of serious intellectual impairment and deterioration that we call dementia. If events continue as predicted, the number of our people in nursing homes will swell dramatically, and 50 to 75% of nursing home residents will have some intellectual impairment. Having been brought up with a grandparent who had severe Alzheimer's disease and lived at home, I am aware that the tragedy of this disease extends beyond mere statistics. It is a disease which affects the very basic human nature of the patient and family.

Dementia in our elderly patient population has been called the silent epidemic. All involved in the health care of the elderly must plan the diagnostic, therapeutic, administrative and research strategies to combat this disease. Dementia is a disease process. We recognize that there is some intellectual deterioration that probably is related to the aging process itself. Yet we also know that many of the great thinkers, leaders, scientists have been elderly people who belie the view that intellectual deterioration in the elderly is inevitable. Dementia is an abnormal deterioration in intellectual capacity that affects language, memory, personality and knowledge. All elderly patients who experience mental deterioration that affects day to day living need medical evaluation.

Alzheimer's disease is the most common cause of such pathologic decline in intellectual function in the elderly. A patient can be supported through this disease, but at present there is no cure for it.

Before a diagnosis of Alzheimer's is made, we must be sure that no reversible cause of the dementia can be found. No matter how many elderly patients with mental deterioration there may be in the United States at any given time, we must guarantee that all are thoroughly evaluated for the cause of their intellectual impairment. For many studies, and our own experience, have shown that a percentage of older patients with dementia (perhaps 10-25%) will have a disease for which the dementia can be reversed. These patients can be returned to a life of normality with proper diagnosis and therapy. Such diseases as hypothyroidism, certain vitamin deficiency, infections of the nervous system, pernicious anemia and brain tumors can cause mental deterioration in the elderly, but be treated effectively after diagnosis. It is, therefore, recommended that all patients with dementia have a complete physical examination as well as a series of laboratory tests including blood count, thyroid function, urinalysis, chest x-ray, blood chemistries, CAT scan and lumbar puncture. It must also be recognized that elderly patients with depression, hearing loss and medication side effects can be misdiagnosed as dementia.

If no reversible cause for mental deterioration can be found, Alzheimer's disease becomes a most likely diagnosis. There are no definitive tests to prove this disease, although at autopsy there are classic pathologic findings in the brain and the severity of these abnormalities by microscopic study correlate well with the severity of the mental impairment. A work group established by the National Institute of Neurologic and Communicative Disorders has recently established clinical criteria for the diagnosis of Alzheimer's disease.

Once the diagnosis of Alzheimer's disease is made, the goals of our health-care team are to make the patient's life as comfortable and meaningful as possible, maintaining an optimistic approach while recognizing that some degree of continued mental deterioration is inevitable. Whether the patient is cared for at home or in an institutional setting, the emphasis is on a multidisciplinary approach to care, recognizing the patient is as much an individual as he was prior to his disease. The patient in an institutional setting should be seen by social worker, internist, psychiatrist, rehabilitation therapist and nurse specialist. The social worker must help preserve the patient's ties to his family and help the family deal with issues of guilt and rejection that occur with institutionalization. A psychiatrist can help asses the possibility of depression as a concomitant illness. Psychotropic medication may be prescribed.

The internist must come to realize that his patient is a fragile one, that the life span of a patient with Alzheimer's disease is greatly diminished. One reason for the high mortality rate of the elderly patient with Alzheimer's disease is that the patient is less likely to report symptoms of serious illness. The chest pain of heart disease or shortness of breath of pneumonia may go unreported and unnoticed.

Pneumonia is the most common cause of death in the Alzheimer's patient. The Alzheimer's disease patient then, much like the young child, needs very special attention.

Those with training in occupational or arts therapy may help the patient adjust to his declining mental capabilities. Hobbies, interests, pasttimes must adapt to these limitations. In that regard, the development of programs for the patient with Alzheimer's disease must take into account regional differences. The patient who has spent his entire life in the hills of Tennessee might not do as well in support programs designed for a patient from New York. Many Alzheimer's disease patients have an urge to wander. Institutional confinement is likely more severe a restriction to a patient who has lived on a farm than one previously confined to an apartment or tenament.

Last but not least among the health-care team, the well-trained nurse becomes the patient's greatest friend. The quality of a day-to-day existence where the little things are all that are left, depend on trained, compassionate nursing care.

Ideally, an institutionalized setting should adapt to the patient's level of deteriorating function. At the Mountain Home VAMC we have a domicillary, nursing home care unit, intermediate care unit and acute care hospital. all of which are available for the care of the Alzheimer disease patient. A patient with dementia may need to go to levels of more intense care as his disease progresses. A patient with early Alzheimer's disease may function well in a domicillary setting, caring for himself and going to a cafeteria to get his meals. This independence should be preserved as long as possible. However, as memory and judgement fail, a more structured environment such as our nursing home care unit becomes necessary. As a patient loses all ability to communicate his needs or requires constant medical or nursing attention, an intermediate care unit with a full-time physician becomes most appropriate. The facilities of an acute medical facility may be necessary to deal with emergency or life threatening illnesses.

The importance of research into senile dementia cannot be underestimated. It does not appear that a simple solution to Alzheimer's is on the horizon. The view of the disease as a primary acetylcholine deficiency which can be-reversed by dietary choline, lecithin or parenteral physostigmine supplementation does not appear as promising as previously hoped. Data which suggested the disease may be secondary to aluminum toxicity\has not been corroborated and epidemiologic studies argue against it. The theory of Alzheimer's disease as a slow virus infection is intriguing in light of increasing evidence that some slowly dementing diseases (Jacob Creuzfeld, Kuru) are caused by viral infections, and by evidence that the immune system of elderly patients does weaken with age, leaving the elderly susceptible to some infections. Our own research has emphasized the elderly patient's susceptibility to pneumonia and other bacterial infections.

There is no single theory or approach to Alzheimer's disease that is so promising as to preclude funding for original or different points of view. We are a long distance from a cure for Alzheimer's disease and the best path has not yet been determined. In that regard we will likely be supporting and caring for patients with Alzheimer's disease for many years. Physicians and basic scientists will be the focus of most research support. However, it is possible that research into methods to best provide quality of life for the Alzheimer's disease patient and family will be equally important. Home health care and innovative institutionalized settings are attractive areas for research support. Nursing homes must be established which become centers for research and teaching about the problems of Alzheimer's disease and aging in general. The clinical scientist who works with elderly patients, assesses their daily bedside problems but also has training in clinical investigation is the physician most likely to make significant contributions to patient care.

At the Veterans Administration Medical Center and ETSU College of Medicine, we hope to develop the best possible approach to the care of our dementia patients, to teach and inspire our students to become physicians sensitive to the challenges of aging, and to use our unique resources to contribute to the understanding of this disease.

I would like to thank the committee for giving me this opportunity to describe the problem of Alzheimer's disease as seen through the eyes of an internist.

STEVEN L. BERK, M.D.
Associate Professor and Chief
Division of Infectious Disease
East Tennessee State University
Quillen-Dishner College of Medicine
Chief of Medicine
Veterans Administration Medical Center
Mountain Home, Tennessee

Born - March 12, 1949, New York City

Education

Brandeis University - BA - 1971
Waltham, Mass.
Phi Beta Kappa
Honors in Anthropology

Boston University School of Medicine - MD - 1975
Research Fellow in Muscle Disease

Medical Experience

1982-	Chief of Medicine Veterans Administration Medical Center Johnson City, TN
1979-1983	Chief, Division of Infectious Disease Veterans Administration Medical Center Johnson City, TN
1977-1979	Research and Clinical Fellow in Infectious Diseases, Boston City Hospital
1977-1978	Senior Medical Resident Boston City Hospital
1976-1977	Medical Resident Boston City Hospital
1975-1976	Medical Intern Boston City Hospital
1975	Extern, Indian Health Service Keams Canyon, Arizona

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Teaching Experience

1982	Chief, Infectious Disease Section East Tennessee State University Quillen-Dishner College of Medicine Johnson City, TN

1982	Director Residency Training Program (28 Medical Residents) Veterans Administration Medical Center Johnson City, TN
1981	Associate Professor of Medicine Associate Chief of Infectious Disease East Tennessee State University Quillen-Dishner College of Medicine Johnson City, TN
1979-1980	Assistant Professor of Medicine East Tennessee State University Quillen-Dishner College of Medicine Johnson City, TN
1977-1979	Teaching Fellow in Medicine Preceptor in Physical Diagnosis Boston University School of Medicine

Certification

Board Certified Internal Medicine 1979
Board Certified Infectious Disease 1982

Membership

Association of Program Directors in Internal Medicine

Association of Veterans Administration Chiefs of Medicine

Tennessee Society of Clinical Microbiology

Tennessee Lung Association

East Tennessee State University President's Trust

American Geriatric Society

Publications

Approximately 30 publications including book chapters on Infections in Elderly Patients

Awards

Teacher of the year, East Tennessee State University Quillen-Dishner College of Medicine, 1984

Teaching Award presented by Graduating Class at Commencement 1982, 1983, 1984

East Tennessee State University, Quillen-Dishner College of Medicine

Chairman, Research and Publications Committee - Department of Medicine, 1982 - 1983

Chairman, Physical Diagnosis ICM II Course, Second Year Students

Member, Curriculum Committee, ETSU College of Medicine

Chairman, Curriculum Committee, Department of Medicine

Coordinator, Fourth Year Elective in Infectious Disease and Microbiology

Member, Institutional Review Board, East Tennessee State University, 1982

Member, Student Promotions Committee

Coordinator, 3rd and 4th Year Medical Clerkship

Veterans Administration Medical Center

Chief, Medical Service

Chairman, Utilization Review Committee

Member, Pharmacy and Therapeutic Committee

Chief of Medicine Representative to District Planning Board

Coordinator, Geriatric Mini-Residency Program

Mr. GORE. Thank you very much for your statement. We appreciate that. [Applause.]

Mr. GORE. I'm going to call on Senator Pepper for questions.

Mr. PEPPER. Thank you, Mr. Chairman. This panel, I'm sure you'll agree, simply adds to the excellence of this hearing that we've had today. I've attended many hearings in many parts of the country over many years; I just don't see how panels could have been better than you've been here today in the presentation of the facts and the actualities, practicabilities, of this challenging situation.

As you began by pointing out, Dr. Fox, this problem, is increasing in importance and demand upon our country, because it's prevalent among the elderly population, and the elderly population is the growing percentage of the total population. So as you point out, the group above 85 is the fastest growing group of the elderly in the country percentagewise, and those are people particularly vulnerable to this disease. We are dealing with an increasing problem and challenge to us, not a diminishing one. So, that simply emphasizes the necessity of our making all possible speed in our research and in our discovery of new means and measures by which we can help these tragically afflicted people.

Now, I was very much interested in one figure that Mr. Stephens, pointing out that already the deductible in Medicare is $356. That means if any person in this audience has to go to a hospital for a serious illness, you've got to pay $356. That's your so-called deductible.

You pointed out how some people had to go back into a nursing home under Medicaid because they couldn't pay the $356. I consider that relevant to the recommendations made recently by the Advisory Commission on Social Security, which proposed as a solution to the Medicare financial problem comparable to the financial problem we had with respect to Social Security in 1982 and 1983. They said the way to solve the Medicare problem of financial shortage in 4 years, make the people pay more. You said there are a great many can't even pay what we have to pay today.

And then they said, "Let's increase the age of eligibility from 65 to 67."

What are those poor people who can't pay high hospital and doctors' fees—what are they going to do between 65 and 67 if that's the remedy? I don't think Congress is going to adopt that approach to meeting the financial problems of Medicare.

And last, Dr. Berk, now you're representing the Veterans' Administration which means so much to the veterans of our country. And yet, even the Veterans' Administration is not prepared to deal with this problem. Mr. Gore has pointed out that there are 400,000 veterans who are the victims of Alzheimer's disease.

You don't have a real long-care program, do you, for taking care of any veteran that has a long-care problem?

Mr. Gore and I, as Members of Congress, are constantly and pathetically approached by wives or members of the families of veterans, who've said, "Please don't let them turn the veteran out of the hospital. We've got nowhere to put him and he's got to be cared for." Some plaintive wife will be saying, "I can't take care of him, please don't let them send him home."

Well, we called the Veterans' Administration, which is always sympathetic, but those beds in the veterans' hospitals are for sick veterans who have some illness that can be aided, generally, by care, by treatment. They're not long-term care beds where you stay in it the rest of your life just to be cared for, treated as best they can for an incurable disease. So, you can see the magnitude of this problem. We've even considered the possibility—and I introduced in the Congress a resolution—to provide a bipartisan commission to make the long-term study of this whole problem of long-term care, including examining the possibility of putting together Medicaid and Medicare, maybe even putting together civilian treatment and the Veterans' Administration. I don't want to deprive a veteran of a thing. I just don't know what is the best way to deal with it. But we do know we need long-term care solutions to these tragic problems. The veterans need it as well as the civilians of the country need it.

So, you blaze the way, showing us the increasing necessity of the problem. It's a challenge, now, that you give to the Congress and the country to try to move toward meeting that challenge. I commend you in the warmest way for the excellence of your presentation.

Thank you, Mr. Chairman.

Mr. GORE. Thank you, Mr. Pepper.

Let me ask you if you would consent to additional questions in writing that the subcommittee would provide, if that's all right. Let me just ask for a couple of very brief questions since we've gone over our time.

First of all, Ms. Carper, you stated that in your study dealing with the families of Alzheimer's victims, you asked them about their experience when the doctor told them that their family member had Alzheimer's disease; 72 percent of them said that the explanation given was poor or nonexistent, and then 75 percent of them reported that the physician never gave them any advice at all about what to expect or how to cope with the disease, even though the family members were going to have to live with it for maybe 12 to 15 years—maybe less, maybe more—24 hours a day.

I said earlier that I think the main reason for that is that the medical community, just like all of the rest of us in this country, are only recently becoming aware of what this is all about. But I wanted to ask Dr. Fox for his reaction to your study.

Because, Dr. Fox, your statement, frankly, struck me as very sensitive and competent. I was very, very impressed with what you had to say, and I think it's indicative of the talent assembled at this medical school. But I'm sure you were concerned, as I was, at the results of Ms. Carper's study. And what would your reaction be to it?

Dr. Fox. I was concerned but I wasn't surprised. In fact, I think that a large number of physicians don't know what causes—even the current literature on what causes—the condition. Many charts will still see the term "senile" on the chart; which is, again—you know my feelings about that. So, we are talking about a disease that is coming to the fore recently, even though it's been known about for a long time.

We're also talking about a system that's sort of been inadequate in terms of dealing with these problems of the aged for a long time, and a great many doctors in practice who, with all the best of motives, intentions, and good will simply have not come across this piece of literature about medical practice and have been content and successful in practicing in the way they've always practiced regarding this particular kind of problem. It's going to take considerable effort to get that turned around.

Mr. **Gore**. Well, I appreciate your statement.

You at the medical school, and you, Dr. Berk, at the Veterans' Administration at Mountain Home, have begun a very innovative program. And I want to make certain that Senator Pepper knows that this is one of the very few places in the country where an identified population of Alzheimer's patients is being dealt with by a combined effort of the medical school and the Veterans' Administration in an effort to develop better and more innovative programs for providing support, for providing care, and for conducting research. And when the decision is made, hopefully when our legislation is passed and these centers are located, that is an extremely significant point to be considered when the location of these centers is decided.

And of course, your statement and your support, Senator Pepper, will probably be more influential than anything else.

But I wanted to also ask just briefly, in this final question for Mrs. Lancaster and Dr. Berk, have you considered involving veterans groups as part of the effort to develop support mechanisms here in your program?

Mrs. **Lancaster**. The only thing that I have done for the support group is that I have contacted all the veterans groups in the area, trying to elicit their support for basically just contacting the people in the community who are victims—or the families of the victims.

Mr. **Gore**. Well, that's a resource of dedication and commitment that I think we want to look more at. As a member of the VFW, myself, and the American Legion, I think that this has some possibilities.

Let me make just a few statements, and then we're going to conclude this hearing.

Mr. **Pepper**. Mr. Chairman, let me, just before you make your——

Mr. **Gore**. Yes.

Mr. **Pepper** [continuing]. Final, concluding statement, let me just say that it's very interesting and very gratifying to me to find you, here in this relatively small institution, your relatively small veterans assembly, you ladies with your organizations to help, that you've done so much innovative work. It's a wonderful tribute to the quality of people that you have here as well as your conscience. And I commend all of you, you who have come forward, coming up with innovative and sympathetic programs of help for the people as well as the aging, what you are doing here in your Aging Commission. I commend you upon the enterprise and initiative that you all are showing, giving good leadership to the Nation in what you are doing. Thank you.

Mr. **Gore**. Thank you, Senator Pepper.

Let me conclude by saying that there are copies of all of the witnesses statements on the table over here to the left. If you've heard a witness that you wanted to read up on, you wanted another copy nor a copy of the statement, we have some limited copies of statements over on the table to the left.

A reminder that we're going to have sign-up sheets here on the witness table for those who would like to get additional information about Alzheimer's disease and about assistance for families and support groups, et cetera.

Another reminder: There will be a reception at 2 o'clock for those who would want to meet Senator Pepper, in rooms 6 and 7 across the hall.

Now, in conclusion I would like to thank all of the witnesses who have appeared here today. I would like to thank the East Tennessee State University and the medical school for their tremendous hospitality in making these arrangements available. I would like to thank our audience. We had more than 500 people here today who attended this hearing, and I hope the information provided is valuable.

I would like to remind you all that this is the first of 3 days of hearings, and we are going to continue an intensive effort to try to better coordinate our Nation's response to this challenge.

And finally, I'd like to add, in conclusion, a special word of thanks to Senator Claude Pepper who—you have seen for yourselves—is such an undaunted and magnificent champion for Americans of all ages and particularly older Americans, for being here at this hearing today.

To all of you, thank you very, very much.

[Applause.]

[Whereupon, at 1:19 p.m., the hearing was concluded.]

ALZHEIMER'S DISEASE RESEARCH

WEDNESDAY, SEPTEMBER 19, 1984

House of Representatives,
Committee on Science and Technology,
Subcommittee on Investigations and Oversight,
Washington, DC.

The subcommittee met, pursuant to call, at 9:30 a.m.; in room 2325, Rayburn House Office Building, Hon. Albert Gore, Jr. (chairman of the subcommittee) presiding.

Mr. Gore. The subcommittee will come to order.

I would like to welcome all of our witnesses and guests.

Today we begin the second of 3 days of hearings on Alzheimer's disease. The first day was held in Johnson City, TN on August 30. The next day will be tomorrow. Tomorrow we look forward to hearing from the two scientists who are responsible for the two recent breakthroughs announced in the press over the last few weeks.

In the first day of this series of hearings, we heard testimony that included many startling facts about Alzheimer's disease. First, there is a 40-percent chance that the diagnosis of Alzheimer's disease is incorrect.

Second, there is no cure.

Third, there is no effective treatment, only maintenance.

The cost for maintenance is around $30,000 per year per patient.

There is little financial assistance from governmental agencies.

The disease lasts from a few years to two decades.

Eventually, the victim loses all intellectual ability.

Seventy-five percent of Alzheimer's caregivers reported receiving no advice from physicians.

All of these facts lead to the conclusion that for the victim and caregivers, Alzheimer's disease is a nightmare that continues for years.

Alzheimer's is not a new disease. It was first described in 1906, but since then there has been little real progress in understanding the causes and in developing effective treatments.

It was for years confused with senility, as if senility was some natural result of old age. We now know, however, that is not the case.

Recently, however, new scientific research results have been reported that may help unlock some of the secrets. Several of the witnesses will discuss these new findings.

Alzheimer's disease is not rare. As many as 10 percent of people over the age of 65 are victims. The aging population in the United States is increasing rapidly and, accordingly, the number of Alzheimer's victims is also expected to increase from 2 million today to 4 million by the turn of the century.

On the other hand, it does strike younger age groups—the youngest victim was 28—but the frequency of occurrence is much less.

Compounding the problem is the lack of a consistent Federal policy in health care for Alzheimer's victims. For example, long-term health care is not adequately covered by Medicare or Medicaid. Compounding this problem is the fact that the Veterans' Administration does not have a consistent policy. In the coming years this could be very significant because by the year 2000, two-thirds of all American males over the age of 65 will be veterans.

This progressive debilitating disease must be conquered. In order to conquer it, we must have an outstanding research effort along with adequate health care programs.

Over the next 2 days, witnesses will testify on the status of the latest scientific research into the causes, cures, and treatments of Alzheimer's. In addition, we will examine the future Federal research program needs and explore the various options in health care funding.

Now, I would like to at this point recognize my distinguished colleague and friend from Kansas, the ranking minority member of the full Science and Technology Committee, Mr. Larry Winn, Jr., without whose assistance these hearings would not have taken place.

Let me also say that Congressman Winn is retiring at the end of this Congress, and I would like to take this opportunity, as I have on other occasions, to say how much we are going to miss him, and I am delighted and pleased that we have had this opportunity to work closely together on an issue as important as Alzheimer's disease.

Mr. WINN. Thank you very much, Mr. Chairman.

I want to compliment you for holding these 2 days of hearings here in Washington on this important topic. You made our witnesses and guests here today aware that this subcommittee has already held 1 day of hearings on this subject several weeks ago in your home State of Tennessee.

I expressed my opinion to you that this topic was sufficiently important and of such national concern that several days of hearings here in Washington would also be beneficial.

I am pleased that we were able to work things out to schedule these 2 days prior to the end of the 98th Congress.

Over the next 2 days we will be hearing from numerous witnesses and panels on suggested causes, research, and effects of Alzheimer's disease. All of these people have a great deal more expertise in their subject fields than I, so I will not take up the subcommittee's time with any extensive overview of the problem or a layman's view of these complex issues and research.

Let me just say that Alzheimer's disease is a relatively new disease—as far as making an impression on the Nation's consciousness. However, those of us who have witnessed the tragic dimensions of this disease as it has impacted on our own family and loved ones realize that it is a serious and far-reaching illness.

The Nation and, in particular, the medical community must give increased attention to Alzheimer's in order that we may control and hopefully eventually eliminate the disease.

I know that we here in Congress are ready to help in any reasonable way that we can. We manifested our awareness and our concerns about Alzheimer's by passing a measure which I cosponsored, House Joint Resolution 451. President Reagan has signed that measure into Public Law 98-310.

With that, Mr. Chairman, let me say welcome to all of our witnesses today. I look forward to the next 2 days of hearings with a great deal of interest and with the expectation that the subcommittee will shortly be able to publish a far-reaching and helpful report on Alzheimer's disease.

I thank you, Mr. Chairman.

Mr. Gore. Thank you.

The ranking minority member of this subcommittee, a very distinguished colleague and friend, Mr. Skeen, is recognized at this time.

Mr. Skeen. Thank you, Mr. Chairman.

I have no statement. I just want to commend you and Congressman Winn for holding these hearings. I think this issue is of significant importance to warrant such an extensive hearing schedule.

I welcome the witnesses today. And would ask unanimous consent to have a statement entered in the record from Congressman Carney, who could not be here this morning.

Mr. Gore. Without objection, that will certainly be done.

[The opening statement of Mr. Carney follows:]

Opening Statement by Hon. William Carney

Thank you, Mr. Chairman, and good morning.

I want to commend you and Congressman Winn for calling these important hearings. Alzheimer's disease is truly a disease of devastating proportions affecting both the victim and care-giver alike.

There are approximately 2.5 million middle-aged and elderly individuals affected by Alzheimer's disease in this country. And, as the elderly population of the United States continues to increase, the number of individuals with Alzheimer's disease is also expected to grow. For example, while in 1978 there were about 2.5 million people 85 years of age or older, by the year 2000 the population over 85 may reach 6,700,000.

Recently, breakthroughs in scientific research have improved our understanding of what causes Alzheimer's disease and how to treat it successfully. At the present time, research is the one best hope for victims and families. Since 1976, Alzheimer's disease has become a major priority for private and publically supported scientific research. Federal support of research on Alzheimer's disease has increased from less than $4 million in 1976 to approximately $25 million in 1983. Scientific studies of the diagnosis, causes and course of Alzheimer's disease have now established a firm foundation for research on the care and possible treatment of its victims.

I am confident that at some future date, we may be able to cure or even prevent Alzheimer's disease. If we do, it will only be with the continued support and cooperation of Congress, the Federal research agencies, the medical community, and support groups of the victims and care-givers of Alzheimer's disease that we will hear from in these two days of hearings.

Thank you, Mr. Chairman.

Mr. Gore. Congresswoman Schneider.

Mrs. Schneider. No comments.

Mr. Gore. Let's go right to our first witness. We are pleased and honored to have Senator John Heinz lead off the hearing.

Senator Heinz is chairman of the Special Committee on Aging in the Senate and has devoted a good deal of time to this problem. We are delighted to have you here.

STATEMENT OF HON. JOHN HEINZ, A U.S. SENATOR FROM THE STATE OF PENNSYLVANIA

Senator HEINZ. Thank you very much, Mr. Chairman.

Before I begin, Senator Pressler had intended to be here. He has a conflict.

He has asked that I ask the committee to put his full statement in the record.

Mr. GORE. We would be delighted to. Without objection his statement will be included at this point.

[The prepared statement of Senator Pressler follows:]

TESTIMONY BY SENATOR LARRY PRESSLER

Mr. Chairman, I thank you for the opportunity to testify today. I have a personal interest in this topic but, more importantly, I am very concerned about the millions of Americans who could face the tragedies of Alzheimer's Disease. I salute you for holding this hearing and increasing public awareness on this issue. Each time the House or Senate takes time to address this issue I believe we take a big step forward in our effort to find answers.

The fact that Alzheimer's Disease will strike one out of every six parents carries special meaning for me. My father was diagnosed as having the disease several years ago. The shock we all felt at the time has worn off, but I am afraid that what will never end is the pain and sorrow of seeing an otherwise healthy, loving, sharp-witted person deprived of the joy that the so-called "golden years" are supposed to represent.

Alzheimer's Disease is afflicting more than 2.5 million Americans. The National Institute on Aging estimates that one-half of the nursing home patients today suffer from Alzheimer's Disease. The costs of caring for these individuals and those at home now exceed $26 billion annually. This is a bad situation which could grow much worse as greater numbers of our population enter the higher age brackets. The experts predict that in 40 years there will be 43 million Americans age 65 and over—or nearly double today's elderly population. This is the age group that is most vulnerable to Alzheimer's Disease.

Unless we are able to solve the mysteries surrounding this disease, we can expect a serious hemorrhaging of our society in the not too distant future. The answers must come through research—high-quality, sustained research—and the full backing of the federal government.

I have been working toward that goal since 1982 when I was successful in attaching an amendment to the Continuing Resolution requiring the National Institute on Aging to spend no less than $6.5 million for research of Alzheimer's Disease. Unfortunately, that amendment was dropped in conference. I then co-authored a letter to the Director of the National Institutes of Health urging greater priority to research on Alzheimer's Disease. I also have addressed the need for thorough, high-quality research at hearings before Senate and House committees.

At my request, two hearings of the Senate Special Committee on Aging were held in the fall of 1983. The first hearing was held in my home state of South Dakota. That hearing brought to the forefront the shortcomings of geriatrics training and the need for better communication between those doing research and those giving care. The Alzheimer's Disease and Related Disorders Association is taking the lead in making a manual available to the many care-givers working with Alzheimer's victims.

At a second hearing held at the Jewish Home and Hospital for Aged in New York City, Senator Heinz and I learned how important it is for us not only to fund research but to address the difficult questions of financing long-term care for those afflicted with this tragic ailment.

After that hearing, Senator Heinz, Senator D'Amato and I wrote a letter to the Chairman of the Appropriations Subcommittee on Labor, HHS and Education urging the addition of $15 million for basic research. Due to that action, the appropriation bill included $23 million for Alzheimer's Disease and $3.5 million for five research centers specializing in this research.

Difficult questions before us include not only the causes which you address here today, but also the long-term implications in dealing with the demand for long-term and acute care due to the rise in prevalence. It has become vitally necessary for us to assess alternatives to nursing home care—including home, day, respite and domiciliary care. Those of us in Congress will have to address the options for financing long-term care, including the possibility of long-term care insurance. This issue will be addressed in a Senate Aging Committee hearing this Friday.

I believe that it is most important for us to continue to provide a forum for addressing the many problems we encounter because of the advent of Alzheimer's Disease and other related neurological disorders. I salute you for taking a look at possible scientific causes. You may be assured of my help in our common cause of finding answers to the many issues that this disease has brought to the forefront.

Senator HEINZ. I thank the Chair. I commend the Chair. I am quite honored to be here.

I commend the chairman and the ranking minority member, Larry Winn of Kansas, with whom I had the privilege to serve for so many years in this, the other body. What you have done, I think, in scheduling these two days of hearings is to focus not only a spotlight on Alzheimer's, but to put it under the microscope figuratively and literally as well.

It would be my hope, with yours, Mr. Chairman, that as a result of this that we will all gain greater knowledge of how we can and should continue to make much more progress and understanding of this tragic disease.

I would note that today the Congress will receive the report by the Department of Health and Human Services task force on Alzheimer's disease. This task force, which was established by Secretary Heckler last year, has pooled our Nation's scientific and medical resources to produce a coordinated, comprehensive study on Alzheimer's disease.

And I want to take this opportunity to especially commend Secretary Heckler and the Department of Health and Human Services for her leadership, their leadership in this area. I am sure that report will be quite valuable to us all.

As is well known, there are at least 2 million and perhaps as many as 5 million persons, age 65 and over, afflicted with Alzheimer's disease. It kills over 100,000 people a year. But as each of us here in this room has probably known someone with Alzheimer's disease.

Those statistics that I just cited do note even begin to convey for a split second the depth of pain and frustration and family tragedy Alzheimer's afflicts on its victims and his or her loved ones.

Mr. Chairman, I am here in part today, certainly not here to testify like the other witnesses on technical matters as to research—I am here as an advocate. I am here to ask that you join with us in the Senate, as I think you have done on many other occasions, in launching what I would describe as a three-front attack to combat this disease that crushes mind and body and spirit.

I would like to suggest that, of course, our first line of attack involves what you are doing here. It should be to expand significantly our current research efforts, expand them because we all know we have only scratched the surface in understanding this dreadful disease, and part of the problem has been in the past inadequate funds.

In that connection, during last year's Senate Aging Committee hearings on Alzheimer's disease, we in the Aging Committee learned that we are spending only $1 on Alzheimer's research for every $2,000 we spend on care for the victims. All the more significant when you realize that not a lot of care from institutions, from home health aids, is given to Alzheimer's victims—probably 75 to 80 percent of all that is given to Alzheimer's victims is given, so to speak, free of charge by the family of the victim.

I think that the $1 dollar we spend on research to the $2,000 that we incur in costs is a clear example of penny wise but pound foolish spending, and common sense would tell us that increases in research funding beyond what is currently contemplated will help accelerate our finding the cause and cure for this disease.

And it seems especially important that such funding be a dependable commitment to major annual increases. If we do that, Mr. Chairman, I think we will have made a commitment that is consistent both with our instinct for compassion and for savings to the taxpayer.

In his opening statement, Chairman Gore and also Larry Winn, mentioned the word "caregivers." And there are two major initiatives, I believe, at a minimum, we need to take with respect to care-giving.

The first has to do with the total inadequacy of our institutional ability to deliver care to the elderly. It has often bothered me, for example, that although we have some 425 medical—425 teaching hospitals associated with some 127 medical schools, that only 15 of those 127 medical schools requires students to take courses in geriatric care, No. 1, and that only 6 of those 127 medical schools affiliated with the 127 hospitals are affiliated with teaching nursing homes—only 7.

We have numerous statistics drawn from the recent study done by the National Institute on Aging that indicates that as we look ahead toward 1990, we will need 10 times the number of geriatric physicians that we have today. That is only 6 years from now.

We will need a fivefold increase in geriatric medicine faculty, going from 275 to 1,350. We will need a doubling of the number of registered nurses with at least some training in geriatrics.

We will need a substantial increase in geriatric nurse faculty, obviously, to accommodate that. We will need a sixfold increase in geriatric dentistry faculty, a doubling of geriatric community health nurses, substantial increase in geriatric social workers; an increase in the general social work faculty.

Indeed, we not only, as I have cited, need the people who will be professional caregivers, but we need the people to train them and we don't even have the people to train them now.

It is for that reason that the Committee on Aging has held hearings on this subject. Clearly, it is an important element if we are going to care properly for our elderly, including those who have always, and it is for that reason, in order to estabish a second front on Alzheimer's and other problems, health problems of the elderly, that I and some of my colleagues in the Senate are today introducing my bill, the Geriatric Manpower Act of 1984.

I don't expect that bill to go anyplace in 1984. But I do ask you to take a look at it for what we need to do in 1985. And I do ask that you join with us on this Senate Committee on Aging in that regard.

The third line of attack, and perhaps our greatest challenge, as we all know, is to improve access and affordability of long-term care. As I mentioned a moment ago, victims of Alzheimer's disease and most people who have long-term care needs get most of that care from their families.

There are not many alternatives. And, as you have heard, I am sure, on previous occasions, the family of an Alzheimer's person ultimately may face probably in the majority of cases what is called the spend-down problem. That kind of minimizes the problem.

What it really means is that a formerly self-supporting couple, husband and wife, are going to find themselves at some point having to place the ill member of their family in a nursing home at $25,000 to $30,000 a year—if they have $70,000 or $80,000 savings, within a couple of years that is gone.

The only way you can keep it up is to go on welfare, sell your house or get divorced. And that is an absolutely terrible choice for our society to have to inflict on two people who have loved each other all their lives.

A small help would be for—and I suspect a good deal more in the way of hope—would be to alleviate some of the burden of such families, is exemplified by a bill that I have introduced, together with others, S. 1301.

It is a bill to provide a tax credit for expenses incurred in the care at home of family members who suffer from Alzheimer's disease. I urge the members of the House to give this bill serious consideration. And anything you can do to move it ahead would be deeply appreciated.

Let me just finally, Mr. Chairman, take this occasion to note that the Special Committee on Aging will have a hearing this Friday to look at the cost of long-term care for patients and their families.

This committee will consider options to further alleviate this financial burden, such as long-term care insurance, private sector or public/private sector insurance. We look forward to continuing to work with our House and our Senate colleagues to enact legislation that makes home and community-based services available to the frail elderly.

In conclusion, Mr. Chairman, I commend this subcommittee for their hearing. What you are doing will help bring about more timely, more decisive action, not only to increase our understanding of Alzheimer's, but to conquer that disease.

Thank you very much.

Mr. GORE. Thank you. We certainly appreciate your being here.

I want to recognize my colleagues for questions at this time. But before doing that, I want to recognize Mr. Carney for any opening statement that he cares to make.

Mr. CARNEY. Thank you, Mr. Chairman.

I believe a unanimous consent request was made that my opening statement be submitted for the record. I appreciate that.

I perhaps maybe would yield to the question at this point. If I could make a statement after Senator Heinz was asked the questions, because I am sure he has to get back to the other side.

I would not want to hold him up.

Mr. GORE. Fine. Mr. Winn.

Mr. WINN. Thank you, Mr. Chairman.

I think the chairman and I are both aware of the report that is pending in the next day or so—I believe it may come out today—from Health and Human Services, and the interest has been expressed by Mrs. Heckler, Secretary Heckler, in the subject matter.

As a matter of fact, I personally asked her if she would appear and could reveal some contents of that report at this meeting, but other plans had been made before that.

Anyway, this subcommittee and the full committee will have benefit of that report and I appreciate your bringing that to the attention of the committee.

I want to commend you and the Senate Committee on Aging for the work that you have done in this field. It makes me feel very good that you are going to be in the next Congress, serving in the other body, and that you can continue your good work.

Senator HEINZ. Don't thank me. Thank the Constitution.

Mr. WINN. Well, the Constitution worked out well. And possibly my friend from Tennessee may be joining you in that body.

Senator HEINZ. I notice he did not forcefully gavel you down.

Mr. WINN. No. Very quitely.

Mr. GORE. That was a simultaneous knock on wood, as well as a gavel.

Mr. WINN. The work that you have done, I think, and the statistical information that you have developed in your committee is going to be very, very helpful, certainly, in persuading the Members of Congress in the future years of the tremendous need for care of Alzheimer's patients, and the fact that you have pointed out—you made a very good point that most of the present load is carried by the families, and in many cases we have seen and heard of and read about some very touching human stories of what the disease has done to families and their loved ones.

I am looking forward to helping in any way I can, although as the chairman has pointed out, I won't be here in 1985—I still hope I have some friends back here where I can be helpful—on your geriatrics bill of 1984.

As you know, and I know, and as you stated, Congress is running out of time, and we will not be able to get to your bill in this session of Congress. S. 1301, the tax credit, I am a little familiar with that, and I hope that we can do a better job on the House developing a companion bill, which will come to the attention of the various committees of the House in 1985.

I don't have any questions. I just want to urge you, Senator, to keep up your good work. We have a lot of work to do.

I sort of feel like we are the underdogs, as they say in sports. This disease is sort of late blooming as far as publicity and the public is concerned, but possibly we can play catchup and be very helpful.

And I am looking forward to the Senate and to the House leading the way.

I have no futher questions, Mr. Chairman.

Mr. GORE. Mr. Skeen.

Congresswoman Schneider.

Mrs. SCHNEIDER. Senator Heinz, it is always a treat to have the opportunity to work with you and especially to have you before our committee on this topic, which we are quite well aware of your fine reputation.

I am delighted to see that you have come up with a solution that we have been talking about for a long while now, and that is the aging population and having adequate geriatric support to accommodate this aging population.

I know that your leadership as a member of the Clearinghouse on the Future, where we both work together, we have had predictions of the jobs of the future and there have been many studies indicating there are great employment opportunities in the field of geriatrics.

So I congratulate you on coming up with your Geriatric Manpower Act of 1984, and I am going to be most anxious to see that we move such a thing here in the House. But I would be a little more anxious to hear about some of the specifics that are included in that legislation.

Senator HEINZ. You name it, we have it in the legislation. And if you have any suggestions, since there will be time to improve the legislation between now and the next Congress, I would welcome it.

I will send you a copy of the bill as soon as it is presented.

Mrs. SCHNEIDER. Thank you. Later this morning we will be getting into some of the various aspects surrounding the problems of Alzheimer's disease. But I am particularly anxious to know from your perspective what role the Federal Government can play in solving some of the problems of cost for the families.

You mentioned the idea of tax credit for some of the expenses in the home. I think that you recognize as well as the rest of us there is much discussion coming forward now as to what we are going to do next year with our tax system.

And there are proposals for flat rate taxes making it very clear that any plans to have tax credits, tax deductions are really going to be minimal.

I would just like to have some of your comments on this. I think there might be an alternative strategy, if we do go the flat rate proposal.

Senator HEINZ. I suspect it is unlikely that Congress is going to dramatically overhaul the entire Tax Code next year. It will be, if we ever decide to do it, an enormously complex task and we are not going to get it done in the first 6 months of next year, and everybody knows we are going to have to address the elements of the budget in the first 6 months.

While there may not be any tax increases, we understand that there are going to be at least at a minimum some substantial revenue enhancement.

Having said that, I would be the first to say that tax credits are offered as much a combination of psychological support as financial support for current care-givers, as they will financially help.

But I think many people tend to get discouraged. They need, for example, some respite care, so that they can physically carry on. These are the family care-givers I am talking about.

If you can buy a little respite through a tax credit for the care giver, you have really helped in many ways far beyond the money involved.

But I cannot sit here and predict whether we are going to have a flat tax or a fair and reasonable tax or a VAT tax. But the longer term answer here is that we have to fashion a national strategy involving long-term care that makes—that is affordable, that makes some sense, and that fits into our current mind set against new Federal programs.

Now, the life insurance industry has had a great financial success over decades, over generations, selling debt insurance, insurance against debt as life insurance. I suspect they would not be too successful selling debilitating disease insurance, always insurance.

Everybody says that that is not going to happen to me, and when it does happen to you, you may not realize that it did happen to you.

I want to challenge our insurance industry to develop independent living insurance, something everybody wants to do, wants to be, independent, and it is my hope that we will lay a considerable amount of ground work for that at our hearing this Friday, which is why I took the occasion to announce that hearing today.

Mrs. SCHNEIDER. How about insofar as the Federal Government is concerned in covering some costs, such as in home health care. Is that a possibility, or is that ongoing now? I am really not aware as to whether visiting nurses are covered under Medicare or whatever.

Senator HEINZ. Unfortunately, there has been some retreat on present policy in that regard. It used to be possible following a period of hospitalization for—or a period of convalescence in a nursing home to get what in practice amounted to as much home health care as you needed as long as you were getting better.

Under new regulations, that has been reduced substantially and may, if my memory serves me correctly, be limited to about 30 days—a 30-day period during which you may receive home health care.

That is not much time, even if you are convalescing. So I would have to tell you in all honesty we are not moving ahead in that area.

We seem to be slipping back on a slope.

Mrs. SCHNEIDER. One final question I would like to ask you. I found it rather interesting back in my home State of Rhode Island just this past weekend there has been some discussion about having a National Health Insurance Program.

I think we, the realists here in Washington—or, at least, we try to be realistic—have acknowledged that the likelihood of a National Health Insurance Program is pretty bleak—even Senator Kennedy himself had introduced legislation and since that time, withdrawn it, recognizing that he just could not deliver on national health care.

However, there are continuing discussions on catastrophic health care. I was wondering if you felt that the care for Alzheimer's disease might be something that ought to be specifically named in any legislation that would provide catastrophic health care.

Senator HEINZ. Two comments on that. The first is that it may very well be that Alzheimer's should be considered, at least in its later stages, an acute care problem which would at a minimum render the afflicted person eligible for the new hospice benefit.

Second, if we ever get to a long-term care strategy, I would think that Alzheimer's should be the first among them, first among equals, if you will, as something that should be protected against.

You mentioned catastrophic, per se. I would direct the attention of interested members of this committee—I recognize it is the Science and Technology Committee—to a bill I introduced some months ago called the Medicare Incentives Reform Act, which includes in it a catastrophic coverge benefit.

But I caution you—it is in the frame work of Medicare. And our definition of what is catastrophic is you have to have an acute illness from which you can recover or have a reasonable prospect of recovering.

That is the typical definition we use as to what constitutes a catastrophic illness, as opposed to a humanly catastrophic illness, which is certainly the case with Alzheimer's and what one might think a commonsense interpretation of the term might be.

I would only mention, I guess for Congressman Gore's benefit, the way we finance our catastrophic coverage is through doubling the excise tax on cigarettes. But I am sure that won't pose any problems for you.

Mr. **Gore**. Mr. Carney.

Mr. **Carney**. Thank you very much, Senator.

I thank you for coming before the committee. You pointed out something that I think is very, very important, and that is that we are spending a dollar in research for every $2,000 in health care.

If my memory serves me, going back several years to when I was very young, when my family was struck by tuberculosis, and we had to send two of my aunts away for extended health care, and we spent money on research.

What brought the cost down in the area of TB was quite simply the fact that we found a prevention to tuberculosis. And I think this is something we have to do, and particularly we members of the panel of Science and Technology have to focus on research and development, because the cheapest way we can deal with this terrible disease is to find a preventive method so the disease does not reach the proportions that tuberculosis did, if it has not already, and become perhaps the most expensive disease in our country to deal with in the future. The statistics point to it is becoming that, as was so ably brought out in your testimony.

Along those lines, I would urge that we make sure we maintain a double thrust. Certainly aid to those who are caregivers is imperative today, because we don't have the solutions. But we would not be utilizing our Government's best ability and our industries' best ability if we were not spending more money on research and development to try to find out more about the disease.

Along those lines, when we look at we have only spent a dollar for every $2,000, and quite honestly, a shamefully small amount of money for research, and we look at some of the breakthroughs that we recognized in the past couple of years dealing with this disease, it seems to me that the greater amount of research and development at this particular time is called for.

I would like to bring your attention to a program that is going on in my own congressional district, the Brook Haven National Laboratory, dealing with the positron emission tomography. They have a two-phased program—doing research on Alzheimer's disease.

The first phase has already been completed, where they catalog, you might want to say for the lack of a better term, the brain's metabolic function with the progression of the disease.

And we have learned an awful lot as far as what is happening with the disease. And now we are in the second phase, and that is the phase of utilizing some drug therapy. And we have had breakthroughs there—not significant, but we have seen whereby utilizing drug therapy, we have either halted the progression of the disease or had recognized some remission of the disease, short-lived though it may be, but to me these are signs that are telling us, invest some money, invest some money—because there is enough knowledge to give us a direction to spend our research dollars on as far as this disease is concerned.

And if we can make that major type of breakthrough that we have done so many other times in this Nation, when it comes to disease, we would have answered the problem in a more thorough fashion.

I don't mean to say, Senator, that your concerns for caregivers are not well directed. I share them with you. But I would hope that we don't take money from research to give it to caregivers or vice versa.

We have to do both. We have to recognize the necessity to do both. And then and only then will we find the answers to a problem that I think could become the most dreaded disease in America, particularly when we look at our demographics, the age of our population as it becomes an older and older population—this disease obviously is one that affects our elderly more so than our youth.

We could almost chart numbers of people who will be victims of this disease in the future, and those charts are frightening. It goes off on a level that would make it virtually impossible for people to deal with, the cost associated with caring for those.

So we have to make an investment today. And it would be, I believe, a very, very prudent investment on Government's part.

The National Institutes of Health is doing that. And I would hope that in the 99th Congress that we, Members of both Houses, would support the NIH endeavors in this to a far greater level than we are today.

Then and only then will we get to the basic problem here, and hopefully in the next 10, 20 years—I would like to be optimistic and say 5 years, we will have enough information to eradicate the spread of the disease. And that is what my thrust was.

But I want to again compliment you for everything that you have done, particularly your emphasis on the caregivers and the enormous impact it has on people who have members of their family that have suffered from this disease.

It just totally wipes out families' financial resources in a very short period of time. It is a dreaded disease that we have to work on immediately.

Senator HEINZ. Congressman, thank you very much. I totally agree with you that we have to do both the usual kind of research as well as improve our ability to train the people to give care.

And you are quite correct that many of the remarkable advances that seem to be on the horizon are coming from quite spectacular technology like tomography, and I gather, for example, there has been some identification of how the—the overproduction of one enzyme can lead to some of the problems of Alzheimer's which I am sure you will hear about from people who know something about it rather than me.

But I want to make one point. There is an enormous amount of emphasis traditionally on the kind of research via project submissions and RFP's that is reviewed by peer groups, and that kind of research, project research, where a very smart guy writes up a project, sends it in, it is reviewed by a lot of other very smart guys, and they say that is terrific, fund it, do it, we get a lot from that.

It is very important to do that. That is what we think of most of the time when we talk about health care research. That is what NIH does most of the time.

That is not, however, I suggest, the only mode for making discoveries in the scientific area. And we are going to find that there are cross-disciplinary approaches involving people who are much more in tune with what is going on at the patient level, and that those interdisciplinary and cross disciplinary and multilayered disciplinary approaches can always yield results, hence, the suggestion—the reason I suggested to you the importance of improving the faculty of the people who train geriatric caregivers was not simply out of compassion or care, but directly related to how research discoveries are made.

We have funded last year, I guess, five—again, this year another five—Alzheimer's centers for research. Those centers—excuse me—Alzheimer's centers. Those centers are partly care giving centers.

But they are also important to research, because they are hands on, not just pure laboratory, not just using some marvellous 10-mile wide device that bends subatomic particles around a higher and higher energy.

And so having trained people on faculty, people who are teaching, people doing research in order to teach people is a very important part of the research effort on Alzheimer's and other diseases of this kind.

Mr. Carney. I think the caution we have to watch is that we don't compete between health care——

Senator Heinz. No argument. I agree entirely.

Mr. Carney. I just hope your colleagues would recognize we have to go forward with a dual thrust to address this problem.

Thank you.

Mr. Gore. Thank you, Senator Heinz. We appreciate your appearance here today.

Senator Heinz. Mr. Chairman, thank you, members of the committee, I appreciate the chance to be here. I just want to recognize my colleague, the chairman of the House Committee on Aging, Ed Roybal.

Ed, good morning, nice to see you.

Mr. Roybal. Mr. Chairman, before the Senator leaves.

Mr. Gore. I was just about to recognize our distinguished colleague, the chairman of the Select Committee on Aging who is going to make the next statement. If you would care to ask some questions.

Mr. ROYBAL. I have been with Senator Heinz on various panels. I know how well he answers questions.

I am not going to ask him any questions at this time. I simply want to congratulate him and thank him for the work that he has done in the other body, in the Senate of the United States, on behalf of the elderly, but particularly on Alzheimer's disease.

I always want to congratulate and thank the chairman of this committee and the members of this committee for having the foresight, first of all, of calling this hearing, focusing on this tremendous problem.

You made reference, Senator, to the fact that money was appropriated in 1984. That is correct. I made the amendment in the Committee on Appropriations of which I am a member. We were able to get $3.5 million.

This year the motion was for $5 million for Alzheimer's disease research. We have a problem. And one of the reasons that I wanted to come to greet you this morning is that we passed the legislation in the House.

The Senate has not. We would like the Senate to pass that same piece of legislation, and to go to conference to see if we cannot put into law those recommendations that have been made—many of these recommendations would affect research on Alzheimer's, health and education, and so forth.

If that is not done, we will have to then pass a continuing resolution. At this moment that bill is not in the continuing resolution. But if there is no action taken by the Senate, it will have to be put in, and it is my understanding that it will be sometime this coming Friday that the continuing resolution will pass in the House.

I am bringing that to your attention because you have been most cooperative, we have worked very closely together, and I hope that we can continue this effort. See what you can do on the Senate side.

I realize what the situation is. But maybe just a little push from you would result in the passing of that legislation. The President has agreed to sign it.

I would rather see a bill signed by the President than having to run this country on a continuing resolution as we have for the last 5 years.

Senator HEINZ. Chairman Roybal, let me agree with you a thousand times over. It has been some frustration to all of us that the Labor-HHS appropriation always seems to end up either a day late or on occasion a dollar short.

We will do all we can to further expeditious consideration of that in the Senate. I know you know that there are a number of other considerations having nothing to do with the Labor-HHS appropriation.

Maybe you and I should be on one of the defense committees.

Mr. ROYBAL. I understand what you are saying. Thank you very much.

Mr. GORE. Thank you, Senator Heinz.

We have a quorum on the floor. We are going to continue this hearing.

Mr. GORE. I would like to now call our first panel of witnesses. Some of us will go over to the floor, and Congressman Winn will take over the Chair of the hearing.

I would like to call Mrs. Virginia Burns of Overland Park, KS; Mr. Clyde Shugart of Perry Hall, MD; and Mrs. Nadine Grashot of Memphis, TN.

We would be delighted to hear your testimony at this time.

I know that all of you have very important statements to make. I want to welcome all three of you.

I hope my colleagues will forgive me if I extend a very special welcome to Mrs. Grashot, because I know her and have visited the center that she has established in Memphis, TN.

We are going to hear first from Mrs. Burns, then from Mr. Shugart, and then Mrs. Grashot. Mrs. Burns, for your statement I am going to turn over the gavel to Mr. Winn. Those of us who are going over to the floor will return very shortly.

Mr. WINN [presiding]. Thank you very much, Mr. Chairman.

I ask that we continue to hear this panel of three witnesses, because it looks like we may be running a shade late anyway. I will give up my right to vote just to say that I am there ready to do business.

The other members will be back. So why don't we continue right now, Mrs. Burns. You have a written statement. If you want to submit the whole statement or present it in brief, however you want to handle it.

STATEMENTS OF VIRGINIA BURNS, CLYDE SHUGART, AND NADINE GRASHOT, ALZHEIMER'S DAY CARE CENTER, MEMPHIS, TN

Mrs. BURNS. Well, I would like to take the written statement and add a little to it.

I would like to thank you for the opportunity to be here today. My name is Virginia Burns. My husband, Joe, has Alzheimer's.

When I first started speaking with Mr. Lamina about my appearing today, I had hoped to bring my husband with me. I really feel until you have met a person with Alzheimer's personally, that you cannot really understand the effect that it has. After checking with the health advisors in our Alzheimer's organization, I was advised it would not be in his best interests to come.

Also, I was a little selfish because we have not been separated in ten years, and I miss having him with me.

We Live in Overland Park, KS, which is a suburb of Kansas City. We have lived there for 24 years. Our three adult children no longer are at home. Joe is a electrical engineer.

He had worked for 28 years for AT&T before his illness struck. His disease was diagnosed in 1974. Prior to that time we had noticed little changes in Joe's ability to cope and in his personality. He would get in the car and start to go to work and realize he forgot his keys, or he would call me from the office and say where is my billfold, I cannot seem to find it. He loved to play chess and enjoyed winning.

He would take his lunch with him and play on his lunch hour. But he came home saying, "Those people sure are getting better at chess because I am losing more often." I said, "You are getting old, you cannot expect to win all the time."

He began having difficulty getting all of his work done and was bringing it home with him to complete. He felt he was understaffed.

He was sent to a company-sponsored school at a university and it was a course that he himself had taught, and he had attended many times. But he could not seem to understand what the teacher was trying to teach.

His defense was, well, he must be a poor teacher. One weekend he happened to bring some of the work home and showed it to our high school aged son and myself. We understood what he was talking about.

We could always find an excuse if he was having difficulties. We took him to a psychiatrist, because we felt maybe this was job related stress. After the psychiatrist spoke with him for a little while, that is what he decided it was.

But it wasn't very long after that until he was told by his superiors his job performance was not up to par. And this really upset him, because he had always enjoyed a very good job rating.

So we went back to the psychiatrist, who did some outpatient testing, and nothing was wrong, everything turned out normal.

In November of 1974 we were celebrating our 25th wedding anniversary. Our children wanted to give us an anniversary party. And Joe did not feel he could cope with this.

To eliminate that possibility we left town so there could not be a surprise party. Shortly after we returned, he was admitted to the hospital for a complete neurological examination.

After he had been in the hospital for a few days, I called the doctor and I said, "Have you reached a diagnosis yet?" And he said, "Meet me at the hospital tonight, I would like to tell the two of you together." And that didn't sound good to me.

But I met him and it was at that time that he told the two of us together that Joe had pre-senile dementia. He said he could return to work, but he would strongly suggest early retirement.

Joe was not emotionally or financially able to even consider retirement at this time. I was allowed to take Joe home and we spent a great deal of time crying together, and holding each other and trying to console each other and plan for a very uncertain future.

Our children could not understand how this could happen to their father. He had been such a capable father. And we still had two children in college and this really put the future of their education in doubt.

We were called to Joe's office when he recovered enough from the tests which made him very ill and were told by—we met with the company doctor and with his superiors. We were told at that time he would not be allowed to come back to work, that he must take disability retirement. And this had not been in our plans.

He was put on disability salary for 1 year. After that time he was put on disability pension and continues to be on that. We applied for and received social security disability.

All we really knew about the disease is that it would eventually be fatal. Basically, I was just told to take him home and wait for him to die.

I had never been employed fulltime outside of the house. But by the end of January I had a job and was starting to work.

The first day that I left, Joe was sobbing and saying why couldn't he be working because he wanted very much to be working and for me to stay home where I belonged. After that we had complete role reversal.

He was staying home doing what he could. Sometimes he would have dinner ready when I came home. And I was working.

Every day when I would come home I would expect to find him dead sitting in his chair, because I had no idea what this disease was to do to him. I was not even aware it was not a circulatory disease.

It wasn't until a year later when I asked our doctor for a letter for the IRS for permanent disability that the term Alzheimer's was applied to his illness. I would like to think the reason I was not given a complete description or more accurate description of the disease is because the physicians themselves did not know 10 years ago.

We were told that it was extremely rare. I went 6½ years thinking Joe was the only one in Kansas City with this disease. I felt very guilty.

We didn't know the reason for the disease. I felt I must have caused it in some way.

During this time the illness progressed. When I would call Joe during the day to check on him, he would no longer answer the telephone. I would leave his lunch for him. He would forget to eat.

There was one time I was reasonably sure that he had eaten cat food I had left in the refrigerator. The time was coming that I must make the decision whether I was going to continue to work and place him in a nursing home or whether I should quit my job and stay home and care for him.

My love and concern for my husband was greater than my desire for a career, so I did quit, and I stayed home with him fulltime.

Things began to kind of close in on me. I became depressed. I felt our lives were over. There was no future. And I can well remember the day that I made an appointment with our family doctor and told him of my thoughts of suicide, taking Joe with me.

There was no other way. I know now that because of my desire to live, my deep religious feelings, that I had no thought of ever carrying this thought through. But I was reaching for help that was not available to me.

We no longer had a social life. People did not understand. I did not understand.

So I began counseling with our local mental health and it was through this counsellor that I was put in touch with a social worker who was thinking about starting an Alzheimer's support group.

I can remember my first phone call to her. It was an hour long, very tearful conversation. This was the first time I had spoken with anyone who gave me any information about the disease, and let me know that I was not the only one with this problem.

My first mistake was in telling her I would do anything to make this group a working group. And I have been very active in the group from that day on.

At last there was someone who understood. I was given written information about the disease, and it was more horrible than I had anticipated. Now, 3½ years later, this has made the difference to me of life and death.

I was offered no hope, but I was given the support and understanding that I needed.

Joe's personality changed during this time. He went from a very strong person who was capable of taking care of himself to someone who needs constant care.

He was losing his ability to express himself. Almost any question asked him he would reply, "You better believe it, baby," and this was completely out of character for him.

He began to lose control of his kidneys. This made life outside of our home a real problem. There has to be a funny side to everything. I would like to share this with you.

We took a 1-day trip. I decided he probably needed to go to the bathroom. So I stopped at a roadside rest area, waited very patiently outside the ladies' room until there was no one in there, and I took him in, hoping we would achieve the required results very fast.

Well, naturally, while we were in there, everybody came in. There were a lot of women in there. So in my haste to hurry him out of there, I guess I kind of disturbed him, and in his very deep male voice he said, "Oh, baby, oh, baby." So there was no doubt there was a man in the ladies' room.

As with all families, there were problems that I would like to have discussed with him. But he no longer understands anything.

There is no reason to discuss things with him. Everything is left up to me. If we receive any correspondence that I would like to share with him, this no longer can be done.

At this time Joe has completely lost his ability to speak and all control of his body functions. He makes a parakeet type of noise constantly, sometimes lasting for hours. We assume this is his form of communication.

I am a widow with a husband. I am a social leper. Our children no longer have a father.

The body that was their father still exists, but the loving, caring father is no longer there. Our daughter was married in November 1979 and we had a small formal church wedding, and he was able to walk her down the aisle, sobbing very loudly all the way down the aisle.

I am sure it was emotional giving his daughter away. But looking back now, I am sure the frustration and not knowing what was expected of him was probably the reason for his sobbing.

Unfortunately, her marriage was not a good one. She was married again in May of this year. This time Joe was in a motel with a hired sitter. Our son gave her away, and I attended the wedding without him.

The first time that I caught myself saying "before Joe died" it was very shocking to me, but I am beginning to feel that way, that he is dead.

Our children have been supportive of me. Without the support, it would have been much more difficult. Due to the fact I have been able to care for Joe at home, we have not had some of the financial difficulties that other couples have had. But if I were to place Joe in a nursing home, I, too, might be faced with bankruptcy.

Nursing home care in our area runs from $1,500 to $2,000 a month and it would not take long to devour our family savings. Due to the fact that Joe was diagnosed at age 50, he was deprived of at least 15 years of productive and financially rewarding years.

We did not have the opportunity to prepare for retirement in the manner we would have liked. Several adult day care centers have opened in our area, but due to location we were not able to use them.

Last April our church opened an adult day care center staffed by hospital personnel. At last I have a place where I can take Joe 1 day a week, get out and do the shopping and things that are necessary to keep our house running.

I know he is well cared for. Before this I would have to leave him in a car, and rush into the grocery store and shop as fast as I could.

This was not pleasant for either of us. And personal shopping was entirely out of the question. I have really enjoyed having this place to take Joe.

But as was mentioned previously, it would be wonderful if we had some financial aid for day care, nursing home care and for those of us who keep our family member at home, because there is a great possibility I will not always be able to take Joe in the day care center.

I really have enjoyed my association with the Alzheimer's group. I have been given understanding and friendship at a time when I really desparately needed it.

I have become concerned for their families. There is great emotional stress when the spouse is advised to divorce their spouse, so that they will no longer be financially responsible for them.

They have family problems that they cannot share with their spouse, so they bring that type of thing to our support group. And when they are faced with putting their loved one in a nursing home, there is a great guilt feeling and when they bring this to the support groups, we can assure them they have our support.

The frustrations, guilt feelings, loss of self-respect, are things that we share and find great comfort in being able to express. I feel the loss of the companionship of my husband.

I have no way of knowing what he feels. He seems somewhat content. He is very willing for me to care for him. And this is a role he would not have previously accepted.

I am sure in the beginning he suffered emotionally. It was just recently that some of the neighbors told me he told them he had been diagnosed as being crazy.

There were misunderstandings. He was very bitter about having been, in his opinion, fired, because he wanted to still be working. There were misunderstandings with relatives, some of which led to permanent breaks with them.

This isn't uncommon because in the early stages of the disease the victim appears to be normal and, therefore, people expect them to act in a normal way. Many families are faced with legal matters.

Some must fight for social security disability. I am pleased that I saw in T.V. last night this is being worked on and revised.

As the person becomes incompetent, the family must decide what is the best way for them to be protected. Some opt guardianship, conservatorship, power of attorney, trust funds, and so forth.

Some of our patients have written bad checks, had car accidents, become confused and lost in public places, are thought of as intoxicated and many have become verbally and physically abusive to family members and others.

I can think of no disease more horrible than Alzheimer's. The patient is not the only victim, but family and friends as well. Many of our caregivers are older as Alzheimer's traditionally strikes the older person and they are not able to physically give the care I am able to give my husband.

But my time may come also. And God forbid that I should become ill. Today at age 57 and 60 our future is still uncertain.

How much longer his living death can continue only God knows. Until the cause and cure can be found, families will continue to be destroyed.

Thank you for the opportunity to be here.

Mr. GORE. Thank you very much for your statement. We will save our questions until all three of you have testified.

[The prepared statement of Mrs. Burns follows:]

TESTIMONY TO THE SUBCOMMITTEE ON INVESTIGATIONS AND OVERSIGHT OF THE SCIENCE AND TECHNOLOGY COMMITTEE - SEPTEMBER 19 & 20, 1984
SUBMITTED BY VIRGINIA L. BURNS

My name is Virginia Burns, wife of Joe Burns, a victim of Alzheimer's Disease. With our three children, we have lived in Overland Park, Kansas, a suburb of Kansas City, Missouri, for the last 24 years. I have been the caregiver for my husband the last ten years. It is because of his illness that I have been asked to testify. I have been an active member of the Greater Kansas City Area Alzheimer's Disease and Related Disorders Association, Inc. I am happy to be able to share with you some of our experiences and those of others with whom I have had contact.

Joe is an electrical engineer, having been employed by A T & T for 28 years before his illness. His disease was diagnosed in December, 1974. Prior to that time we had noticed little changes in his personality and ability to cope. He would forget his keys to the car upon leaving for work. He might call me after he arrived at the office and say "Is my billfold there? I don't have it with me." We would laugh about it and say it must be because he was getting old. He brown paper bagged his lunch, as he loved to play chess on his lunch hour and even more, he loved to win. He began coming home saying that they must be getting better as he was losing more often. Occasionally he would forget to take his lunch. Again, we thought it was old age.

Joe began to have difficulty in getting all of his work done on time and was bringing it home. He was sure he was understaffed. He attended a company sponsored university course out of town. He just couldn't seem to understand what the teacher was teaching. He must be a poor teacher. We could always find an excuse. When things began to close in on him we took Joe to a psychiatrist who, after talking to him thought it was probably job-related stress. It was a few months later that he was told by his superiors that his work was not up to par.

Joe had always enjoyed a good rating and this completely upset him. We again returned to the psychiatrist who put him through out-patient testing. Everything was normal.

At the time of our 25th wedding anniversary, November, 1974, we left town to avoid our children surprising us with an anniversary party. Joe just didn't think he could take that. Shortly after our return, he was admitted to a hospital for a neurological examination. He could no longer add two and two, but we were so sure that it had to be stress and we would just have to take life a little slower. After he had been in the hospital several days, I called the doctor and asked him if he had reached a diagnosis. He asked me to meet him at the hospital that night and he would tell us together. I was sure then that it would not be good. He told us together that Joe had presenile dementia. He said Joe could return to work but he would suggest early retirement. At age 50 Joe was not ready, financially or emotionally, to think about retirement. Of course our lives were devastated. I was allowed to take Joe home and we spent a great amount of time crying together, holding each other and trying to plan for an uncertain future. Our children couldn't understand how this could happen to their father. Two of them were still in college and this certainly left their future education in doubt. We were called to Joe's office and told that due to his illness he would have to take early disability retirement. This had not been in our plans. He was put on disability with full salary for a year. At the end of that time he was put on disability pension. We applied for and received Social Security disability. All we really knew about the disease was that it would eventually be fatal. Basically, I was told to take him home and wait for him to die.

I had never been employed full-time outside of the home. By the end of January, I had a job and started working. The first day when I left, Joe sobbed and said why couldn't he be working and me stay home where I belonged. After that we had complete role reversal. He was staying home doing what he could and I was working. Each day when I came home I really expected to find him dead, as I had no idea what this disease was to do to him.

It wasn't until a year later, when I asked for a letter from the doctor for permanent disability for IRS that the term Alzheimer's was applied to Joe's illness. I like to think the reason I was not given a more complete description of Alzheimer's was because at that time there was not a lot of information available. We were told that it was extremely rare. I continued to believe this for six and a half years. I felt guilt because I knew that I must have caused it in some way. I continued to work for five years. During that time the illness progressed. When I would call Joe during the day to check on him, he would no longer answer. I would leave meals for him and he would forget to eat them. One day I was reasonably sure that he had eaten the cat food which was in the refrigerator. The time had come that I had to make a decision as to whether to quit my job and stay home to care for him or place him in a nursing home. Since my love and concern for him was greater than my desire for a career, the decision was made to quit my job. About this time I became very depressed, feeling our lives were over. I can remember the day I made an appointment with our family doctor and discussed with him the desire to commit suicide, taking Joe with me. I know now that due to my religious feelings and my desire to live, I had no real thought about carrying this out but was reaching for help that was not available. We no longer had a social life. People didn't understand.

I didn't understand either. I began counseling at our local mental health center and it was through my counselor that I was put in touch with a social worker who had been thinking about starting an Alzheimer's support group. My first phone call to her was a tearful hour-long conversation. This was the first time I had spoken with anyone who gave me any information about the disease and made me aware that I was not the only one with this problem. My first mistake was in telling her that I would do anything to make this group a reality. I have been active in the group since that day. At last, someone who understood. I was given written information about the disease. It was more horrible than I had anticipated. Now, three and a half years later, this organization has meant the difference to me of life and death. Without them I could not still be caring for my husband at home. I was offered no hope, but was given the support and understanding that I needed.

Joe's personality had changed during this time. He went from a very strong capable person to someone who needed constant care. He was losing his ability to express himself. Most often when a question was asked of him, his pat answer would be, "You'd better believe it baby.", a phrase he had not previously used. Then he began to lose control of his kidneys. This made life outside of our home a real problem. We became more isolated and lost all contact with former friends and some of our family members. As with all families, there were problems we should have discussed. Now all of these decisions were left to me. He no longer understood. The letters and correspondence with friends could no longer be shared. At this time Joe has completely lost his ability to speak and all control of his bodily functions. He makes a parakeet noise sometimes lasting for hours. I care for him completely as he can do nothing for himself. I am a widow with a husband. A social leper. Our children no longer have a father.

The body that was their father still exists but the loving, caring father is gone. The first time I caught myself saying, "Before Joe died..." I was shocked. Our children have been most supportive of me. Without their support it would have been more difficult.

Due to the fact that I have been able to care for Joe at home, we have not had the financial difficulties, other than loss of earnings, that some couples have. But in the event that I were to have to place Joe in a nursing home, I too, might be looking down the road to bankruptcy as many others have. With nursing home care costing from $1500 to $2000 per month, it doesn't take long to devour the savings of a family. Due to the fact that Joe was diagnosed at age 50 he was denied at least 15 productive and financially rewarding years. We did not have the opportunity to prepare for retirement in the manner we would have liked.

Several adult day care centers have opened in the last few years. Most of them were not practical for our use due to their location. Last April the church to which Joe and I belong opened an adult day care center staffed by personnel from a hospital. At last, I have a place where I can take Joe one day per week and do the shopping and other things necessary to run our home. I know he is well cared for and it gives him an opportunity to see others. Before this, I was leaving him in the car and running, literally, through the store filling my grocery cart as fast as I could. This was not pleasant for him or me. Personal shopping was out of the question.

I have enjoyed my association with the Alzheimer's Disease and Related Disorders Association and have met many wonderful people. Family members and professionals have become my adopted family. They understand and offer help and friendship at a time when I so desperately need them.

I have become as concerned for them and their families as I am for my own. There is great emotional suffering when they are advised to divorce their spouse so that they won't be caught in bankruptcy court. When they have a family problem and no one to share it with, they bring it to our support group. We can't always give answers but we can listen and perhaps let them make their own decisions by sharing them. When they are faced with placing their loved one in a nursing home and have terrible guilt feelings, we can listen and let them know that they have our support. The frustrations, guilt feelings, loss of self respect are things we share and find comfort in being able to express.

I feel a great loss of the companionship of my husband. I have no way of knowing what he may be feeling. He seems somewhat content. He is very willing for me to care for him. This is a role he would not have accepted before. I am sure in the beginning he must have suffered emotionally. It was just a few years ago that I learned from neighbors that he told them that he was crazy. He was bitter at having been "fired". There were misunderstandings with relatives that led to a permanent break with them. This is not uncommon, it is part of the disease. It happens to many families. In the beginning, the Alzheimer's patient appears normal, therefore families expect them to act in a normal manner. Because they cannot act normally, misunderstandings result.

Many of our families are faced with legal matters. Some must fight for Social Security Disability. As the person becomes incompetent families must make the decision as to what is the best way for them to be protected. Some opt for guardianship, conservatorship, power of attorney, trust funds, etc. Some of our patients have written bad checks, had car accidents, become confused and lost in public places, been thought of as intoxicated and some have become verbally and physically abusive to family members and others.

I can think of no disease more horrible than Alzheimer's. The patient is not the only victim but family and friends as well. Many of our caregivers are older, as Alzheimer's traditionally strikes the older person. They cannot physically give the care that I am able to give my husband. That time may come for me also. God forbid that I should become ill.

Today, at ages 57 and 60 our future is still uncertain. How much longer this living death will continue, only God knows. Until the cause and cure can be found, families will continue to be destroyed.

Thank you for the opportunity to share this with you.

MY JOE

There are so many things
That I would like to know
Why him, O God, Why
Please not my Joe.

His mind's so bright and clear
He's charming and he's witty
He's all that I hold dear
He tells me that I'm pretty.

But now he sits and stares
Not seeing anything
He never speaks at all
He can no longer sing.

I bathe him and I feed him
I dress him in his clothes
I hug him and I kiss him
I even wipe his nose.

I miss him so you see
Although he's still around
He no longer knows me
His judgment isn't sound.

Alzheimer's they tell me
He will surely die
Silently I listen
And then begin to cry.

There are so many things
That I would like to know
Why him, O God, Why
Please not my Joe.

Mr. GORE. Our next witness is Mr. Clyde Shugart of Perry Hall, MD. We welcome your statement.

Mr. SHUGART. I am Clyde Shugart from Perry Hall, MD. My wife, Ruth, has Alzheimer's disease.

In the early seventies she began to make mistakes in cooking and things like this. The family began to notice she was having problems.

I took her to our doctor. He examined her and he said, "Well, she just might have a little confusion at this time." It continued, got a little worse.

We took her back to the doctor again. He examined her again and said she acts very much like she might have a brain tumor. So they made arrangements.

She was entered into Greater Baltimore Medical Center on December 13, 1974. She was in there for about a week. At that time they determined she did not have a brain tumor, but they did not know what her problem was.

The doctors were concerned that just previous to that she had started drinking excessively, and they thought maybe this was causing her problem.

When I look back now at the time the doctor came to me and said, "Thank God she doesn't have a brain tumor," I have often thought how lucky we would have been if it had been that, because perhaps she could have been cured at that time.

They moved her directly from GMBC over to Shephard Pratt Hospital, and put her under observation for alcoholism. She stayed there for a period of a little over a month.

During this time she missed her birthday and Christmas and holidays, and two children's birthdays, and she was very upset. At the end of this time they told me she did not have—she was not alcoholic, but her drinking problem was from whatever other problem was, but they could not tell me what it was.

So then the doctors made arrangements to take her to Johns Hopkins Hospital. She was entered in there in March of 1975. They gave her extensive examination and a brain scan and all kinds of tests.

At the end of that period, which I think was 7 days she was in the hospital, they told me that she apparently had some small strokes, so then she was put under the care of a neurologist.

We continued to take her to the neurologist periodically during the next 2 years. Also, I wasn't satisfied with how things were going.

I took her to several other hospitals and different doctors with no results.

Almost 3 years later the neurologist said to me one day, he said, "There is something drastically wrong." He said, "Normally people with strokes stay on an even plane or they improve." He said, "Your wife is deteriorating." He said, "I suggest we put her back in Johns Hopkins."

So they made arrangements. She went back in the Johns Hopkins. And this time they gave her another brain scan and an arterial sclerosis type examination, all types of examination.

She was in there for a period of 12 days. At the end of that time, that is when they told me she had Alzheimer's disease, which I never heard of before, and no idea what it was, and what problems we would have.

Then I took her home. By this time we were having problems with her wandering. I had to have somebody with her part of the time, which she resented very much.

These were extremely hard times, especially between the time that they told me they thought she had a stroke and we got the final diagnosis, because she did so many peculiar things. She was hard to get along with.

The children were very upset. They didn't know what the problem was. I didn't know what the problem was. So then I had to have somebody with her all the time.

And I was still employed and working at that time. In the evenings I would come home and take care of her and on weekends and care for her as such. As we continued along, she continued to lose ground.

One thing led to another. First we would have to put locks on all the cabinet doors, medicine cabinets, even the refrigerator. If we didn't keep everything locked up, she would wander off and we didn't know where she was, and we would have a problem finding her.

One day I came in and found her eating a bar of soap she thought was a candy bar. Another time she got into some other cleaning fluid that made her very sick.

So we had to be very careful with her during this whole period.

Then along in 1972–82, she became very ill, and we had to take her to the hospital and they thought she had pneumonia at that time. But during this time she lost complete ability to swallow.

She lost the use of her ability to walk. And during this time, they told me it would be impossible for me to take her home again.

Well, I felt like we could handle the situation. I had excellent help taking care of her previous to this. We had a hospital bed I talked to the doctors about.

So they finally made arrangements—I had three ladies taking care of her off and on. They made arrangements for the ladies to come over and they told them how to take care of her.

At this time she had to go on a feeding machine, which meant she had to have constant care. They told me, well, if it doesn't work, you just have to put her in a nursing home.

I might regress a little bit on this, because about 4 years previously on the advice of my attorney and doctor, I had made arrangements with a very fine nursing home in case something happened to me suddenly.

I agreed if we couldn't handle it at home, it would be fine. Our family doctor was attentive, and Dr. Peter Rabins from Johns Hopkins came out and saw everything was all right. She had lost complete use of her legs at that time and so she was on a feeding machine 24 hours a day to begin with.

We would have her so many hours on water and so many hours on a special food. Then we would gradually cut back on her food, as she was inactive, and they found out she was gaining weight.

As we cut that, we got to where we would run the feeding machine from like 8 in the morning until 8 at night, and she is still on this procedure. At the present time, she is, of course, completely bedridden.

The only thing she can move at the present time is her head and her right leg a little bit. We try to get her up once a day in a chair, which is becoming more difficult all the time, of course. And we keep her up from 2 to 4 hours, and even during this time we have her on the feeding machine.

She has been unable to talk for something like 5 or 6 years. At the present time she just makes noises. Normally she is very quiet.

But when she yells out, I have asked the doctors what causes this. They think it is muscle spasms. I might mention last night about 3 o'clock she screamed out. I went in there.

I have no way of knowing what hurts. Her hands are very drawn up to her chest now, her legs are drawn up. So I move them. The doctors think it is probably muscle spasms. All I can do is try to guess at what is wrong with her.

People have asked me why I don't put her in a nursing home. Well, I feel like we are giving her excellent care. In fact, the doctors tell me that.

And I have told them at any time they feel that we cannot give her the care that she should receive at home, I would put her in a nursing home. At the present time she has somebody with her almost constantly.

We have a dentist that comes to the house, a podiatrist that comes to the house, the family doctor who will come at most any time we ask him to come, and Dr. Rabins from Johns Hopkins checks her periodically.

In my written report I listed some figures as to the cost last year. These figures I have here is what is entered in my medical expenses, which comes to like $22,500. There is a tremendous number of hidden costs in keeping her at home.

For example, we wash sheets, bedding, and cloths three to five times a day. We use an enormous amount of kleenex, paper towels, and other such things. We have to keep her room very sanitary all the time.

It has been a tremendous burden on the family. I am very fortunate that my children do live pretty close, and do help when it is necessary. And at times on advice of the doctors I have gone away for a period of a week or so, just to get away from it.

And at that time the children will come in and take over. But it has been a tremendous drain. We have no idea how long this will continue, of course.

I will say one thing. I have received some satisfaction from joining the Alzheimer's Disease and Related Disorders Association of Baltimore and Central Maryland. I was treasurer for 2½ years.

I am on the board of directors. And I have appeared on some other panels. I am committed to do anything I can that I think will help in the future to fight this disease.

Thank you.

Mr. GORE. Well, thank you for helping out in our common effort. Our hearts go out to you and to Mrs. Burns for what you are experiencing.

[The prepared statement of Mr. Shugart follows:]

WRITTEN STATEMENT FOR THE HEARING ON ALZHEIMER'S DISEASE BEFORE THE U.S. HOUSE OF REPRESENTATIVES COMMITTEE ON SCIENCE AND TECHNOLOGY SCHEDULED FOR SEPTEMBER 19, 1984 BY CLYDE E. SHUGART

My name is Clyde Shugart and I live at 15 Bangert Avenue, Perry Hall, Maryland 21128. I am employed by High's of Baltimore, Inc., and have been employed by High's since graduating in 1939 from Iowa State University in dairy industry. At the present time I own some stock in the High's corporation. My wife Ruth has Alzheimer's disease and this is my reason for appearing before the Committee.

Some time in the early seventies we began to notice my wife doing some peculiar things. She would make mistakes in cooking, put on clothes that didn't match, misspell words, etc. At that time Ruth was around 54 years of age. Our family doctor thought that she might have a tumor on the brain, so she was admitted to Greater Baltimore Medical Center on December 13, 1974. After extensive examination, it was determined that she did not have a brain tumor. It was thought that perhaps her problems were caused by drinking which she had begun to do in excess.

Ruth was moved directly from G.B.M.C. to Sheppard-Pratt Hospital where she stayed from December 22, 1974 until January 29, 1975. It was determined there that drinking was not the cause of Ruth's problem, but the result of her problem. However, the doctors did not find out what the problem was. By now my wife was having difficulity in talking and in remembering the names of things.

On March 25, 1975 Ruth was entered into Johns Hopkins Hospital where she was given an EMI brain scan and various other tests. At this time they told me that she had apparently had a stroke or maybe several at sometime in the past. Ruth was placed under the care of a neurologist and a speech therapist.

These were extremely trying times as I had to have someone with her at times and she resented this very much. Ruth gave up bowling, garden club, and sometimes was hard to get along with.

I took her to see several different doctors and none of them were of any help. Finally the neurologist said that he thought there was something wrong with her besides having a stroke.

Our family doctor made arrangements for Ruth at Johns Hopkins again and on November 29, 1977 she entered the hospital for 12 more days. She was given another EMI brain scan, a cerebral arteriogram and numerous other tests. It was then determined that Ruth had Alzheimer's Disease, which I had never heard of before.

By this time I had to have someone with my wife at all times. She would wander off and get lost. As time progressed she became more helpless. She lost the use of her right hand and was unable to talk. She became unable to recognize even those closest to her. Eventually we had to feed Ruth, first with food finely chopped and later with a puree. It was increasingly difficult for her to drink liquids.

In the first part of December of 1982, we had to take Ruth to the hospital because it was believed that she had pneumonia. From that time on she has been unable to swallow or walk. Following this trauma, Ruth had to be placed on a feeding machine, tube fed a special formula. She was catheterized as she had become incontinent. Since then, she has been bedridden except that we lift her into a chair so she may be upright for a few hours each day. She is bathed and rotated in bed each day either by myself or another caretaker. A registered nurse comes to see Ruth at least once a week. Our family doctor, Joel Kleinman visits regularly as does Dr. Peter Rabins from Johns Hopkins Hospital.

It is obvious, I am sure, that all of the above has been a tremendous emotional drain on all of our family. The costs of Ruth's care financially are represented by the 1983 figures below.

Special liquid food	$1,889
Equipment rental	$1,440
Feeding equipment	$3,778
Miscellaneous supplies	$ 673
Caretakers	$13,375
Medical expenses (not covered by insurance)	$1,368

The financial costs are not the greatest toll taken by this illness. As mentioned, the emotional drain in terrible: the daily expenditure of time and care-giving leaves us frustrated and feeling hopeless, because we know Ruth will never get well.

One way that I have taken personally to help alieviate this frustration and hopelessness is to participate in The Alzheimer's Disease and Related Disorders Association (ADRDA). Involvment in frequent meetings and lectures, serving on the Board of the local chapter, and acting as the Treasurer for 2½ years has helped me feel like I am contributing in some way. There is nothing I can do directly for Ruth except to provide physical care, but the ADRDA activity may serve to help others with Alzheimer's Disease.

Mr. GORE. We will have some questions for you, but we want to hear from Mrs. Nadine Grashot. Mrs. Grashot is from Memphis, TN, and operates a respite care center, which she can describe much more eloquently than I can. And we would be glad to hear your statement at this time.

Mrs. GRASHOT. Thank you. I am Nadine Grashot, executive director and president of the board of Alzheimer's Day Care, Inc., in Memphis, TN, and the widow of a brilliant, loving man who was a victim of Alzheimer's disease. For these reasons, I have been asked to speak to you this morning on health care for Alzheimer's disease patients in general and the critical need for day care facilities for Alzheimer's victims in particular.

You have read about the clinical effects Alzheimer's disease has on its victims and about the millions of Americans it affects and will hear more on these subjects during these hearings I am sure. My written testimony describes Alzheimer's Day Care, Inc., our daily routine, and our twofold goal of providing quality, dignified care to the patient and counselling and relief from the constant caretaking to the families. You know the facts—now I want to describe the feelings.

If you will, please listen as the wife of one of our participants describes the heartbreak, the loneliness, the desperation of this disease, and the impact that a loving day care can have on a patient and his family:

DEAR NADINE: There are not enough words in my vocabulary to tell you what the Alzheimer's Day Care means to me, as well as to Bob. I do not know how long I can continue to bring him, but for right now it is the best six hours out of our 252 hours a week. (Seven times the 36 hours in an Alzheimer's caretaker's day.)

When I leave him at the day care door and walk out alone I feel like one of Jonathon Livingston's seagulls. Not that I wish to be free of Bob, just free of Alzheimer's Disease. This is a lonely illness—almost of isolation when you can't leave your loved one by himself and can't get any free time to keep up with your friends; they are scared and ashamed of Bob; and they gradually drift away.

As you know, Nadine, the nights are even worse than the days because that is when the patients are more confused. Since Bob has been coming to the day care, we get four to five hours sleep a night where we were getting two to four. That had been going on for two years. He doesn't seem to let his fears or inabilities bother him as much since he comes to the day care. There he is once again in contact with people like him—that can't keep up either.

Although he is still alive, I have already lost my husband to this illness and sometimes I feel I have lost myself—I have no identity. It has been said that everyone needs "me time." In this illness, there is no such word as "me."

I feel when I leave him at the day care you all know how to deal with it and he is safe and I can feel free to do something I want to do or have to do. Before I found the day care, I carried him with me everywhere for the entire four years of his illness.

I drive 35 miles each way to Memphis for this short six hours. If I lived nearer or could afford to come twice a week, he would be there every time you unlocked the door. There is no help from anyone; as you know, this is a living death.

Caretakers truly feel defeated every way they turn, but I know God has seen you and many others through and He will carry us—if we will have the patience to wait for the unfolding of His will for our lives—and if we will only lean on His great strength.

I know the day care is crowded and you need more room and I hope you can get some support and help. Only those who have lived with an Alzheimer's patient know what it is like to try to care for them at home.

Such little decisions as whether the sock goes on before the shoe or the shoe before the sock—even when you get them on right, they still will question whether it is right or not. Sometimes it takes 30 minutes to get his socks and shoes on right. By that time he is ready to pull them off. So, you see, Nadine, for six hours I can let you worry about that. God bless your efforts.

SHIRLEY.

What a difference a day care can make in the remaining years an Alzheimer's patient and his family have together—and what a shame such an option is not available to everyone. A day care can keep Alzheimer's patients out of nursing homes—and off of Government Medicare rolls—longer.

A day care can provide new friendships for patients and their families when their old ones are ashamed or scared and desert them. A day care can help the patient maintain his dignity and self-respect longer.

A day care can help the caretaker maintain the strength, sanity, and sense of humor needed to properly care for the patient. A day care can provide relief to families without funds for private duty nurses but who are not willing to give the patient up to a nursing home.

A day care operating on our five principles of love, compassion, exercise, entertainment, and freedom allows the patient to better cope with the losses and inabilities that he experiences at home. A day care helps preserve the tender feelings between patient and caretaker that Alzheimer's disease and its jealousies and strains try so desperately to tear down.

As the letter says, Alzheimer's disease is a "living death." I know, because I, too, have been there—for 11 long, slow years as my late husband, Louis Grashot, suffered from this fourth leading killer in America.

But there was no day care for us to benefit from. That is why I am doing everything in my power to see that no one ever has to go through this ordeal alone again. I remember thinking one night at 3 a.m. while my husband and I were chasing his "little people" out of the house for the third consecutive night—I remember promising myself not to forget this ordeal, but instead to someday help other victims survive.

I could not leave Louis alone at any time because he would wander off, or pour gasoline in a lawn mower that didn't exist, or try to light an electric stove with matches and paper. Consequently, I could not shop, run errands, or even sleep.

I knew that a day care was the answer and wished with all my heart for one that would provide compassionate love for my husband for just a few hours a day. That is why I and others like me worked so hard to start Alzheimer's Day Care, Inc. 3 months after my husband died, and why I know without any doubt that a day care can do a lot to relieve the trials and anguish of an Alzheimer's patient's family and subsequently the patient himself. A day care can't stop this disease, but it can help.

Alzheimer's Day Care, Inc. in Memphis, TN is a tax-exempt general welfare Tennessee corporation that is acting now, today—trying to help the Alzheimer's patient and his family fulfill his destiny in the dignity and respect that he deserves.

Our program operates 2 days a week in borrowed facilities. We charge a fee of $8 a day, but scholarships are available making our services accessible to everyone. Our patients range in age from 58 to 91, from physically healthy to requiring assistance for walking, from mildly confused to not even knowing when to chew, and from docile to mildly combative.

About 30 percent are incontinent. One patient has forgotten what his own face looks like and is scared of his reflection and chases and fusses at it.

One lady puts food, crayons, or jewelry—hers or anyone else's—in her purse and takes them home. One man sits and hits his leg and face or pulls his hair out if we don't hold and rub his hand and talk soothingly to him.

Some of our patients cry for no apparent reason; some fight and hit and bite; some beg to know "what is wrong with me?" They are all showing signs of severe mental decline, but they are all wonderful human beings who need and get our one-on-one love and attention—not simple babysitting and not restraints or sedation like they might get if treated in a multipurpose day care or nursing home facility unfamiliar with this disease.

We entertain them. We roam with them. We sing and play and do crafts with them. We love them. And we do this with a staff of loyal and dedicated volunteers, most of whom have experienced the Alzheimer's ordeal within their own families. As one caretaker so aptly puts it, "these understanding, caring volunteers are people who have previously walked the path that I am now walking." We have no permanent source of funding, no affiliation with any health or human services agency, and no facilities of our own.

We are a bare beginning of the help that is needed and could be provided with proper and permanent funding, paid assistants, more volunteers, and your help. Our limited space and need for one-on-one supervision allows us to care for only 8 to 10 participants per day.

We have had to turn patients away—some of the patients on our waiting list have died or had to be institutionalized before we could help them. We operate only 12 hours a week because that is the only hours our borrowed facilities are available to us. But if we had larger facilities that were ours and that we could tailor to the specific needs of our participants, we could do so much more.

We could organize separate areas and develop activities appropriate for those in the various stages of Alzheimer's disease with their varying levels of abilities and disabilities. We could have a large enclosed outside area to give the patients fresh air, exercise, freedom, and room to roam in safety.

We could build bathroom facilities designed for the handicapped that are large enough for changing incontinent patients. We could eliminate sharp objects, knives, and other safety hazards.

We could set the hot water thermostat low enough to keep the patients from scalding themselves. We could have non-carpeted, no-wax, nonglare floors that are easy to clean, easy to keep your balance on, and cause no shadows or reflections.

Most importantly, we could give more people more relief from this dreadful disease. Alzheimer's disease affects an estimated 10 to 15 percent of Americans aged 65 and older. With recent Census Bureau studies predicting 35 million people in this age group in the year 2000, America will need to cope with 3.5 to 5.25 million Alzheimer's victims at the turn of the century. We must begin fighting this devastating, widespread disease today.

Thank you for allowing me to make this appeal on behalf of Alzheimer's Day Care, Inc., and I hope this committee will act promptly and favorably toward providing financial assistance and joining us in the fight against the ravages of Alzheimer's.

I personally invite any of you to Memphis to visit our day care operation as Congressman Gore did and see what dedication can do, and then realize how much more can be accomplished with your help and participation.

I thank you.

[The statement of Mrs. Grashot follows:]

THE NEED FOR RESPITE DAY CARE

An Appeal for Patients with Alzheimer's Disease
and Their Families

Alzheimer's Day Care, Inc.
1188 North Parkway
Memphis, Tennessee 38105

Nadine J. Grashot, Executive Director
and President of the Board

Dinah Grashot Allison, B. A.
Board Member

September 5, 1984

THE NEED FOR RESPITE DAY CARE

An Appeal for Patients with Alzheimer's Disease and Their Families

Alzheimer's Disease, recognized as the nation's fourth largest killer, is a progressive, irreversible brain disease with no known cause or cure. While it can strike at any adult age, this is a disease primarily afflicting this country's rapidly growing number of senior citizens. According to Dr. William Applegate, Chief of the Division of Geriatrics in the Department of Community Medicine at the University of Tennessee, ten to fifteen percent of the nation's population over 65 and a larger percentage of those over 85 are victims of Alzheimer's Disease.

When Alzheimer's Disease strikes, it causes its victims to deteriorate to a childlike state usually over a two to ten year period of time. The effort required to take care of an Alzheimer's patient is comparable to that needed to take care of a newborn baby except that the caretaker is not young and strong like a new mother; the patient is not small, loving and learning like a baby; the future is not full of hope and progress like it is for a child. The burden, grief and anguish that this disease inflicts on its patient victims and their families, the often forgotten victims, is indescribable. I know. I have been there.

Because of Alzheimer's Disease, I buried my beloved husband, Louis, in October, 1982. I saw him deteriorate from a loving and devoted husband and father, a successful and intelligent businessman, with a law degree and a former City of Memphis Commissioner of Finance and Institutions, to a person at first confused, then at times frustrated and in various degrees of anxiety and anguish, then disoriented, unable to communicate, unable to find his way out of a room, unable to dress himself, unable to recognize me or our daughter, unable to comprehend the most simple instruction, even incapable of knowing that he should chew or swallow when food was put in his mouth, then on to death.

The English language has no words to describe the emotional and mental anguish he went through in those various stages of the disease and certainly none to describe those experienced by me, our daughter and son-in-law, and the few friends who stood by our side during those years of that long journey to his death. It was heartbreaking and frustrating when many friends and even several family members turned their backs on us and on him because they were embarassed by him or because they could not cope. (They thought we could?). The experience cannot be wiped from my mind nor from my very soul.

It is my belief, as I'm sure it is all of yours, that as a responsible man lives in dignity, he deserves the right to die in dignity. Alzheimer's Disease totally demolishes any such right for that patient. It is maintained only with the help and loving care of the caretaker. After my husband was diagnosed as having Alzheimer's Disease, and while trying to understand and care for him and to cope with my own feelings and sense of desperation, a friend said to me, "Nadine, Louis is going to die. There's nothing you can do about it. Somehow, somewhere, you have to find the courage to watch him do it and you have to find the courage to get through it."

Ladies and gentlemen, the caretaker carries the burden and he or she must survive at the same time in order to be able to carry that burden.

My attempts to survive were made more difficult by the lack of options and resources available.

A home health nurse sent by a doctor said that my husband was not eligible for her services. You see, he charmed her by telling her all sorts of details about his past and about how he managed his daily routine, which, although they were absolutely untrue, convinced her that he or we needed no help.

We called all around town looking for some kind of day care or hospice program that would give some relief from the sleepless nights and the constant supervision, hallucinations, and possessiveness, but we found nothing in the entire Memphis Area.

I couldn't afford around-the-clock nursing attendants because I had to save some money for whatever the future might hold for my husband.

I wasn't ready to give him up to a nursing home.

So, until some of the many physical complications of Alzheimer's Disease put my husband, first in the hospital and later in a nursing home, we managed at home-somehow. Even then, getting this retired U. S. Army Captain, decorated with several medals, into the local Veterans Administration Hospital required the help of Congressman Harold Ford of Memphis, and hospital social workers painted a gloomy picture of the number of nursing homes willing to take Alzheimer's patients.

After his death, in an effort to help Alzheimer's patients and their families, a two day a week respite program was founded in January 1983, was incorporated July 1983 as Alzheimer's Day Care, Inc., and gained tax free status.

It all began with a small group, including myself, who had seen the heartbreaking effects of this disease and who desired to move the concept of a day care from a desired objective for the future to an operative reality.

We found a rent and utility free facility at 1188 North Parkway, Memphis, Tennessee. Memphis Park Commission's Lewis Center for Senior Citizens owns a house next door to their main building and agreed to let the Day Care use the house on Monday and Thursday of each week. Lewis Center also provides hot meals which the patients and volunteers purchase inexpensively, some insurance, volunteer referrals, transportation for some volunteers, limited secretarial support, and much needed moral support.

We found a group of dedicated volunteers willing to donate their time. One volunteer takes one car ride, two buses, and an hour wait to come from as far away as Horn Lake, Mississippi; two volunteers live within the Interstate 40 Loop and ride the Lewis Center bus; one is young and expecting her first child; one only admits to being "75-plus"; there are nurses, housewives, a retired auditor, both men and women; some work full-time and can only give behind-the-scenes support; some are at the Day Care every time the doors are opened. But, they are all loyal and dedicated and treat the patients with the love and dignity they deserve. Most of them have experienced the Alzheimer's ordeal within their own families, and are now willing to contribute their services to ease the stress and provide some brief respite for others who are experiencing the 24-hour responsibility which they endured in the past and vividly remember.

We equipped the facility with an initial expenditure of about $300.00. This was possible because we found generous people to donate furniture and household furnishings, both new and used, as big as a couch and as small as a trash can. We even found a local seafood restaurant willing to contribute a supply of lobster bibs to use when feeding lunch to the patients.

I was drafted as the volunteer director, accepting the operating responsibility for the Day Care and the Day Care became a beautiful reality.

We have a responsible executive board consisting of physicians, nurses, lawyers, business people and a Certified Public Accountant, some of whom are related to past or present Alzheimer's victims but all who are devoted to easing the burden of caretakers of Alzheimer patients and who render a volunteer and faithful service to our organiztion. We have a support group for our families and friends called 'Tender Loving Caretakers' which offers monthly sessions of support and conversational therapy and education. We hope that, by sharing their experiences and little triumphs with each other and with our social worker facilitator, they can better cope with today and tomorrow. Our stated and established goals are:

1. To give the service necessary to allow the caretakers of Alzheimer's some free time and some much needed relief from around-the-clock patient care.
2. To provide the Alzheimer's patient with a safe, supportive and stimulating atmosphere to suit his or her particular capacity.

We believe that these services will enable Alzheimer's patients and their caretakers to lead fuller, more satisfying lives and to better cope with the dibilitating aspects of this disease.

This care is provided at a cost of only $8.00 per day, plus $1.25 for lunch. Since we are currently housed in their facilities, a $5.00 annual membership fee to the Lewis Center for Senior Citizens is also required. Scholarships are available, making the care affordable to all income groups.

Our participants (we prefer this term rather than patients) range in age from 58 to 91, from physically healthy to requiring assistance for walking, from mildly confused to not even knowing when to chew, and from docile to mildly combative. About 30% are incontinent.

Developing activities for participants with such a wide variety of abilities and disabilities is one of our biggest challenges. Group exercises for the participants are difficult to coordinate. A few of the participants make use of exercise bicycles which are available, and there are those who enjoy walking and visiting activities at the Lewis Center for Senior Citizens such as choir rehearsals, band practice, and musical performances. Occasionally these musicians will also perform for the participants at the Day Care and let the participants enjoy a sing-along, even though clapping hands in time with the music may be the limit of participation for some of the participants. The current group of participants do not have the capacity to play cards or dominoes. Although there are participants who are unable to participate in activities, we firmly believe that they derive a benefit from breaking the routine of home with an outing to the Day Care. They all respond to love and kindness, and even those unable to speak have, on occasion, hugged or squeezed the hand of a volunteer. It is then that we know that our efforts are worthwhile.

A pool table next door at the Lewis Center for Senior Citizens is available and provided a favorite pastime for two of the male participants. These two men are particularly interesting because they became close friends and were able to communicate with each other even though they had difficulty communicating with others. They looked forward to their twice weekly get togethers. One is still at the Day Care but has deteriorated to the point that he is no longer able to play pool. The other man is now in a nursing home. The hardest part of volunteering is watching the deterioration of the participants you have loved and cared for - they and their families become like family to us.

Another big challenge was convincing some of the Senior Citizens next door that they could not "catch" Alzheimer's Disease from our participants. They, like so much of the public, have not yet been educated about the facts of this disease. Alzheimer's Disease is a frightening, devastating disease, but it is not contagious.

What is a typical day at Alzheimer's Day Care, Inc. like? As the participants arrive at the Day Care, they are greeted by the one-on-one volunteer assigned to take care of him or her that day. We have to have one volunteer for every participant in order to allow each participant the freedom he needs and deserves while he roams, as all Alzheimer's patients do, and because of the individual help - even help eating, walking, sitting, or going to the bathroom-that each patient needs. The participants see a room decorated with plants they have nurtured and walls decorated by their art work. They start the day with donuts and coffee donated by local businessmen or by participants' families. Morning activities include our own form of exercise, sing-alongs, and walks, followed by an orientation about where we are and what day it is using calendars, addresses, temperatures, etc. Mid morning brings games or toys designed for children, like coloring books, cutouts, building blocks, and Little Golden books. At 11:30 a.m. it is time for lunch, followed by rest time. In the afternoon we might do artwork while we listen to our tape cassette of the Bible or to our children's stories furnished by the Tennessee Library for the Blind and Handicapped. Soon we'll be able to listen to local newspaper excerpts on our WTTL receiver. Normal afternoon refreshments include popcorn, ice cream, cookies, potato chips, and cold drinks. On holidays and participant's birthdays, we celebrate with parties, decorations and special refreshments. At 3:00 p.m., it's time to go home. Our participants will be more content when they go home and may sleep better at night because they have had a busy, active day. Our caretakers, the unsung heroes and heroines that the Day Care seeks to help, will be better able to face their "36-Hour Day"* because they have had some rest and relief.

This the only program of its type geared to the special needs of Alzheimer's patients in Memphis and Shelby County, Tennessee and services some patients from the tri-state area of Arkansas, Mississippi and Tennessee. Our services are provided by a volunteer staff, and one aide furnished by Senior Citizens Services, Inc.; our funds are gathered from donations, nominal participant fees, and small fund-raisers, such as bake sales and carnivals, sponsored by families and friends of Alzheimer's patients; and our facilities and most of our equipment are borrowed. We currently have an active much-needed and well-received program that operates in facilities that Lewis Center for Senior Citizens generously lets us use from 9 a.m. to 3 p.m. on Monday and Thursday.

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*"The 36-Hour Day-A Family Guide to Caring for persons with Alzheimer's Disease, Related Dementing Illnesses, and Memory Loss in Later Life" is the title of a book by Nancy L. Mace and Peter V. Rabins, M. D.

Our facilities allow us to provide respite for only 10 participants at a time.

To date, 31 patients have participated in our program. 10 are now in nursing homes, 2 are hospitalized, 6 have died. However, we know that we are not completely meeting the needs of our current patient families and we are regretfully having to turn away new families in dire need of some support and relief.

The need for a proper and permanent source of funding is indeed great. We need to help more patients but we cannot until we get larger facilities and paid attendants. We need an enviornment that we can tailor to the specific physical and emotional needs of our patients in the various stages of Alzheimer's Disease, including space for the patient to walk without being able to harm himself, but we cannot until we get our own parmanent facilities and needed equipment. We desperately need paid attendants to continue to provide the one-on-one care, supervision, and love that each patient must have. Although they may be mentally and physically impaired, Alzheimer's patients still respond to the tender loving care that this one-on-one relationship allows after they have stopped responding to anything else.

We need to offer our program between eight and nine hours a day, five days a week to allow working caretakers to continue their jobs and to provide more hours of relief to everyone, but we cannot until we get facilities we can use whenever we need them and have the paid attendants to cover those hours.

Remember, ten to fifteen percent of this nation's people over the age 65 are victims of Alzheimer's Disease and this percentage increases with age. According to Congressman Albert Gore, Jr., Alzheimer's Disease has victimized 18,000 people in my home of Shelby County, Tennessee. Since Alzheimer's Day Care, Inc. can only provide respite for 10 participants a day, this leaves 17,990 patients in this one county alone who do not have the opportunity to participate in this kind of program. Typically these patients are either put into nursing homes earlier than they need to be, simply because the family is emotionally and mentally drained, or are kept at home 24 hours a day by a caretaker who becomes physically and/or mentally sick herself because there is no relief. Respite Day Care is the answer for Alzheimer's patients!

We are a program that is acting NOW, TODAY - trying to help the Alzheimer's patient and his family fulfill his destiny in the dignity he deserves. But, we are a bare beginning of the help that is needed and could be provided with proper and permanent funding, paid assistants, more volunteers, and public support.

I personally, invite any and all of you to visit our Day Care and see what we do with these humble beginnings. We need your help and participation.

Thank you for allowing me to make this appeal.

Most Sincerely,

Nadine J. Grashot

Mrs. Nadine J. Grashot, Executive Director
and President of the Board

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BOARD OF DIRECTORS

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Dinah Allison - Senior Information Center Consultant - Federal Express Corporation. Father died with Alzheimer's Disease in October 1982.

Raymond Buring - Vice President of Human Resources - Buring Foods. Nat Buring, father, has Alzheimer's Disease.

Ellie Carper - Administrative Coordinator of Geriatrics at University of Tennessee Community Health Services. Sincere desire to make a contribution and to improve life-style of care-givers.

Nadine Grashot - Director and President of Board - Alzheimer's Day Care, Inc. Husband died with Alzheimer's Disease after 11 years.

Dr. Hollis Halford, Jr. - Director-Department of Nuclear Medicine and Staff Radiologist-Methodist Hospitals. Close personal family friend of Nadine Grashot and her husband, Louis; Mr. Grashot died with Alzheimer's Disease.

Steve Hawks - Treasurer-Alzheimer's Day Care, Inc. Attorney-Incorporating Attorney.

Barbara Lawrence - Secretary-Alzheimer's Day Care, Inc.-Medical Social Worker-Upjohn Health Care Services - assisted in developing the Alzheimer's Day Care at Lewis Center, interested in furthering knowledge of disease.

P. K. Seidman - Vice President - Alzheimer's Day Care, Inc. Attorney and CPA. Alzheimer's Disease of interest due to family patient; also a civic interest.

Mary Simmons - Housewife. Husband is an Alzheimer's victim.

Sue Sydow - Director of Nursing at Allen Morgan Nursing Home-Because of Day Care concept being of such value to patients and families.

Dr. Erika Trapp - Physician

Mr. Gore. Thank you. Thank you for your statement, and for your dedication.

Mr. Winn.

Mr. Winn. I want to thank all three of you for your very touching and personal experiences. I notice there was quite a bit of difference in the attention of the audience for your statements than when the politicians were speaking. Thank you very much for your being here and allowing us to put into the record where we hope to point out to people all over the world what is going on in this field.

Mrs. Burns, I happen to know a little bit about your experiences. You said that it has been—I think all of you said it has been a big financial drain. Mr. Shugart has listed in his prepared remarks, he did not give them orally, the tremendous amount of financial drain that has affected your family, as we know has affected so many others.

So I just wondered if you have any suggestions—and I will ask this question of all of you, if you don't mind, Mr. Chairman—on what you think the Federal Government could do to provide health care assistance.

In your discussions, in your day care centers, your care centers, have you discussed what you think the Federal Government should do? I will start with Mrs. Burns.

Mrs. Burns. No; I really don't think I have ever discussed this with anyone in particular. The fact that Medicare does not pay for what is technically just taking care of them—there has to be improvement for Medicare to pay for this.

As I say, our day care center is $20 a day. That doesn't sound like very much, when I take Joe just 1 day a week. But that is $1,000 a year out of my budget that I had not previously budgeted. Help along this way would be good. I think help with nursing home care would be wonderful.

And also those of us who do have them at home, and I hope to continue keeping Joe at home all the way through, I can see that that is going to become very devastating with the machines, as Mr. Shugart mentioned—the machines you must have to care for them. Any type of help, financial help, that would be available in making their life and my life more comfortable, I would be strongly for.

Mr. Winn. Thank you.

Mr. Shugart.

Mr. Shugart. I think it would be very beneficial if the caregiver at home could get some Government help, especially if we could help keep these people out of nursing homes. I really think they would get better care at home.

But it is a tremendous drain on everybody when they are in nursing homes, there is no question about it. For example, I think my figures will show the feeding machine and special food for that runs over $7,000 a year.

And as I mentioned, there is a lot of hidden costs at home. There is no question that everybody needs help that has an Alzheimer's patient. It is a tremendous financial burden.

But exactly how that should be done, I don't know. But it would certainly help if we could keep people at home as long as possible.

Mr. Winn. Mrs. Grashot.

Mrs. GRASHOT. I thoroughly agree with Mrs. Burns and Mr. Shugart. And, of course, my day care does not apply to some people as advanced probably as their victims. However, we do have them.

As I said in the report, we have patients that are incontinent, and are brought in a wheelchair, cannot walk, and cannot talk. In my opinion, I believe that the day care is truly an answer to a great deal of people's problems, and keeping in mind, too, as we talk about this, there are two victims—there is the victim with the Alzheimer's and there is the victim caretaker.

Never lose sight there are two instead of one victim in Alzheimer's disease. I would like to have the opportunity maybe sometime to present a more complete, thorough listing of what we do. And remember, ours is totally voluntary, and it has been that way for 1 year and 8 months.

So the volunteers can do a lot. We have done it for this long. We could never do an entire program, even with financial help from the Government, which, of course, we would certainly want—even with that, we would still have to have volunteers, because these are the compassionate people, these are the people that give the love and attention to the victim. Paid assistants, yes, they work, and they are good. We need them. But we need them to maybe do some of the work itself, leave some of my volunteers as loving and compassionate as they are, free them up to actually spend an hour a day with no one but that one patient.

So we feel that ours is the answer to a great relief for both victims. And we would like the Federal Government to fund us for that reason.

Mr. WINN. Let me say, and I am sure the chairman would concur, that any additional information that you could get to the committee, I am sure we could include that as a part of your testimony, even though you have not given it.

But if we are trying to produce a comprehensive report, I think some of that information, any information that any of the witnesses have today or tomorrow, we certainly would like to augment your oral statements, particularly based on your experience.

I think most of the members of this committee, and most of the Members of Congress, from time to time have toured day care centers for children or various types of centers of that type, handicapped children, mentally retarded.

I think we are all aware that it is almost a one-on-one as far as voluntary or paid help taking care of those children.

Notice in Alzheimer's, it is basically the same thing. As all of you have stated, you have to watch the patient constantly because of these many things that—many places where they might get into trouble, and might affect their safety or the safety of people around.

So my question is, it seems to me—and I have visted some nursing homes where Alzheimer's patients are, the nursing homes—I don't mean to be critical. I think it is a lack of training, a lack of their help that they have available to them, either paid or voluntary. But they don't have the time to spend with individual patients, and thereby sedation seems to be almost the only answer to them under those circumstances.

And certainly your center, Mrs. Grashot, is doing certainly a better job than that. As you have pointed out in your testimony, sedation is not the answer. That is really, as we all know, to be factual about it, and maybe too harsh, is to calm that patient down and make them sleep or rest. But that is not really helping that patient.

I would like to ask another question, Mr. Chairman, and go down the panel one more time, and then I think we can move on to the other members.

Mrs. Burns, do you have any trouble getting social security?

Mrs. **Burns**. No. We were very fortunate. He was approved without any difficulty at all. But it is not uncommon to be denied social security, and to have to appeal. One of our Alzheimers' organization members just recently went through with that.

The husband was older and she was younger. And they had young children. She had a great deal of difficulty getting disability with social security. But I think last week they had the hearing and it was approved.

We were very fortunate. And we have never been challenged on it. If they ever did, all they would have to do is look at my husband and know he is completely disabled.

Mr. **Winn**. I realize that. But people have been challenged. That is my point. And it is really a problem. On disability insurance, too?

Mrs. **Burns**. Pardon me?

Mr. **Winn**. On disability insurance?

Mrs. **Burns**. I am not sure what you mean by that.

Mr. **Winn**. Any other insurance policy based on disability that he might have purchased during the period of time.

Mrs. **Burns**. We have company insurance at AT&T. It has no coverage at all for Alzheimer's.

Mr. **Winn**. Doesn't mention it one way or the other?

Mrs. **Burns**. No. It is not mentioned by name in any way.

Mr. **Winn**. Mr. Shugart, would you care to address social security?

Mr. **Shugart**. We haven't had any assistance from social security at all. My wife became 65 last December. And then through Medicare, what help we get from them is because I am still employed and under an insurance policy, we have company insurance, that Medicare will pick up some of the difference after the company pays so much of it.

They don't cover all—maybe Medicare will pick up the balance, some of the costs on doctors or special medicines. But there is very little help on medicine, of course.

Our company insurance company does pick up the bulk of that.

Mr. **Winn**. You really have been very fortunate in the fact that you are still and have been all this period of time of your wife's illness a wage earner.

Mr. **Shugart**. Yes. Fortunately, too, I guess it is because I have been with the company so many years, 40-some years, that I really don't put in the hours that I feel like I should, for the simple reason that some days the lady comes 8 in the morning so I don't get to work until about 9. And 2 days a week the lady likes to go home at 5, because she has a family.

And so then I leave early, and the company has been very gracious in the fact that they have ignored that maybe I don't put in the number of hours that people normally do.

But there is other occasions when in an emergency I will. And I have one lady who 3 days a week stays until 6, which makes it convenient for me to stay longer and work on those days.

Mr. **Winn**. Mrs. Grashot, would you care to address the social security problem, because that is one of the things that Senator Heinz talked about. I am sure they are going to look into that.

Mrs. **Grashot**. My husband, Louis, died in October 1982. He was on social security, because at the time he died he was 68 years old. I am 57 years old today. I have no social security. I never have had.

We had absolutely nothing but Louis' check that stopped the day he died. So there is no assistance if you happen to be married to a younger wife. Thank God for my children. I have an ace in the hole.

I have not used them yet, but I will if I have to. And they came with me to this hearing, too.

Mr. **Winn**. Thank you very much for your testimony. As I said, any additional information you care to add, we would be more than glad to have that—in spite of the sad stories that you have told and that we are all aware of, happy birthday.

Mrs. **Grashot**. Thank you.

Mr. **Gore**. Indeed, happy birthday.

Congresswoman Schneider.

Mrs. **Schneider**. I would like to commend all of the members of the panel for their very insightful and moving testimony. I happen to also be a member of the Select Committee on Aging, and so we have had a number of hearings on this very topic.

And I feel that I am quite intimately familiar with the problem that exists. And it is very frustrating to me to be groping towards the solutions and the appropriate solutions.

I would like your assistance in that respect because I think that as we look toward the future, which I am always trying to do, we are going to have innumerable challenges toward coming up with adequate funding and changing the mechanisms, whether it be within medicare or within our tax system for tax credits, or in home health care, or whatever it might be.

Those challenges are great, and the tradeoffs are going to be significant. And I would like to encourage the three of you to be aware of the value of political action, and that means to make it very clear to your representatives, both in the House and the Senate, about the need for some kind of financial and governmental consideration.

We have many different diseases that are all very critical and debilitating. We have many of them that are demanding attention of the Federal Government.

But I think that there is very little question that Alzheimer's disease ought to be put on the top of any priority list for such consideration, not only because of the pain and the extensive care that is required, but also because of the duration of the disease.

I think that these points really need to be made over and over again to your representatives, and certainly, Mrs. Grashot, with your adult day care facility, you have already in place a nationwide mechanism of adult day care centers that certainly is in a position to help communicate this to legislators.

I have been to adult day care centers on numerous occasions in my own State of Rhode Island, and also in Connecticut. As a matter of fact, just on Monday I was visiting one in Connecticut to compare the kind of work they are doing with what we are doing in Rhode Island.

So we need to educate the legislators further and to let them know that this is a critical issue. So I hope that you will become politically involved.

I know oftentimes you have your hands full and you say you don't want to get involved in politics. It seems from the testimony here today that some of the solutions required to help us address the problems certainly are going to be done in the political sector. So I encourage you to do that.

I have no further questions. Thank you.

Mr. Gore. Let me pursue this issue of day care or respite care.

You, and some people helping you, organized this day care center in Memphis, TN. Is it the only center in Memphis that offers this sort of care?

Mrs. Grashot. I don't know of anyone anywhere that is for Alzheimer's patients, not adult day care. We do not believe that the two can be combined, that a mental illness of any other kind and an Alzheimer's patient can be combined successfully.

My people respond to love. There is nothing in the world that they will respond to, or at times, except love, and I mean touch love. When they say something to you, you listen and you say yes or you say no, or you try to, if you catch anything at all that makes sense to what they are saying, talk back to them.

We talk back to them all the time. There is no communication. And that is the reason in this letter to me, the lady said that they are there with their own kind. They can communicate. They go and shoot pool. No one else in the world would shoot pool with them, but my people shoot pool.

It is a way of entertaining on their level. This is important. It could happen to any of us today or tomorrow. Any of us could go into Alzheimer's today, tomorrow, next week.

How would we feel if I were or if our life were taken away from us? We try to give them a place in life and make them feel that they are still somebody. They were, and I, one day—and my husband is a brilliant man.

Of course, I am prejudiced there too. He was, and to see this man, brilliant as he was, go into nothing but a vegetable, is heart breaking. So in there, with the help of Federal aid or something, we can take care of these people.

There are approximately 18,000 people in Memphis, Shelby County, with Alzheimer's disease, and we can only take care of 8 or 10 a day, because we don't have the facilities, the money. All we have got are big working hearts, and we would like to be able to do more, but we are almost at a standstill unless somebody or some agency comes to our help.

Mr. Gore. You may only be taking care of 10, but you have pointed the way to a significant part of a solution for many families who are coping with this illness.

The letter that you read, and the experiences that others have shared with us during our first day of hearings, confirms that even 6 hours a week, 6 to 12 hours a week, offers some relief where you can go shopping and whatnot.

Mrs. Burns, would you agree that at least during the earlier stages of the disease this sort of respite care is a good idea?

Mrs. BURNS. Very good for the patient and the family member. We are socially isolated and if I don't take my husband to a day care center, he doesn't see anybody but me for days and days and days, so it is good for him to be with people of his own kind.

We don't necessarily—ourselves—to Alzheimer's, but it is dementia of some kind, and it is important for him and for me to be there. He is being cared for today with donated time in a nursing home at our area who is specializing at this time in Alzheimer's care.

Mr. GORE. Did you say for a while the only way you could go shopping for groceries was to take your husband with you and lock him in the car and race through the aisles of the grocery store to fill up the cart as quickly as possible?

Mrs. BURNS. That is true, and after finding the car in the middle of a parking lot one day, I had learned to wedge the wheel before I leave him. He had reached over and released the emergency brake, and I found the car in the middle of the parking lot.

So that is true. In the beginning, I could take him in with me, but because of his behavior and his constant noise, it is just best that he not be in a grocery store or a place like that.

Mr. GORE. How do you go about shopping now?

Mrs. BURNS. I take him to the day care center 1 day a week, and I have 7 hours, I can do as I please.

Mr. GORE. I see.

I guess it is a ridiculous understatement to say how much you look forward to those 7 hours?

Mrs. BURNS. You are right, it is. It is wonderful to—well, in the beginning, I was doing a few things that I really had to do. You can't imagine what it is like to do something in the house, repair something, I can't even run up to the hardware store and buy a nail without leaving him in the car.

I make a list on Wednesday, and I even on occasion eat my lunch out. It feels strange to eat without having to cut up somebody else's meat and feed him.

Mr. GORE. Mrs. Grashot, you have discovered in your special program for Alzheimer's patients, that there are some things you do that seem to help, such as vigorous exercise, is that correct?

Mrs. GRASHOT. Yes, we exercise our people. We literally have to take their arms or legs and make them do it. It seems to make them brighter, mentally or physically brighter, more alert for the entire day and then on into—because of the exercise itself, they sleep and rest better that night.

One thing I want to impress upon everybody concerned, and I am sure that I will be—my colleagues will agree with me, is my people need space. They need roaming space. They need to be walked.

We are on a very, very busy street. We really have little business taking our people out for a walk, but we have to, because they need this exercise and fresh air.

We need an area that we can fence in that we can let them go out on their own with no way of getting outside of the fenced-in area, whatever. Let them go out there by themselves, let the people have the freedom. They don't know what freedom is anymore.

It is something in their heads that they constantly fight for, but if they had these things, they would not be so belligerent and antagonistic at home. That is my opinion.

Mr. GORE. Well, as the Congress looks at what the range of responses is to Alzheimer's, certainly respite care has to be included. Your testimony here is very valuable in that respect.

Mrs. Burns, you—before I go on, one other thing. You all engage in something that you call reality orientation?

Mrs. GRASHOT. Yes, we have that every day. We tell—go through a ritual of the address where we are, the Memphis, TN, and without asking the question of, do you know who you are, we go around the room and let everyone tell who they are, if they can.

We try never to ask an Alzheimer's patient a question, because if they can't answer it, they frustrate themselves even more, and so we go into colors. The work with the people that we have, it is mostly on the kindergarten level.

We have blocks and if they know the color, they are so proud of themselves. Also, we go through the alphabet, not through it, but with letters, asking them if they know a word that starts with that letter, and they are just so proud of themselves when they do.

That is reality orientation.

Mr. GORE. Tell them whether it is summer or winter?

Mrs. GRASHOT. We go into the temperature, whether it is going to rain, yes, all sorts of things that you and I take for granted. Today is such-and-such a day and we try to find in the almanac what day it is, Monday, yes, Monday was Citizenship Day, which is a very small thing, but they remember the word "citizenship," one did, and they are very pleased with themselves. They are very pleased when they function.

Mr. GORE. Mrs. Burns, you said in your statement, when you first received your diagnosis, you were not really told anything about what to expect or what you might do to cope or anything like that.

We heard at our first day of hearings, at our first hearing, we received a study, again from Memphis, TN, indicating that 75 percent of Alzheimer's patients' families reported that they received no advice or assistance whatever from the diagnosing physician, but that instead, they were simply told to take the patient home with no instructions, no assistance, and no indication of what was in store for them, even though in some cases, what was in store for them was 10 to 12 years of having the sole responsibility for caring for this very complex illness.

Your own experience was similar to that, is that correct?

Mrs. BURNS. That is correct. The doctor made the statement to me somewhat similar to Mr. Shugart's doctor made, "I wish I could tell it was a malignant tumor, because it is a more kind diagnosis, because it is of short duration."

There was never a time the doctor called me in and spoke with me individually and told me what to expect. I didn't even realize it was not a circulatory problem, because the CAT scan was not available at the time he was diagnosed, but the other testing did prove there was atrophy of the brain.

So, not being familiar with things like this, I assumed it probably was circulatory.

It was not until we formed our Alzheimer's group that I was given any written information as to what the disease was, what to expect to happen, and really, even the duration of it.

Those of us who are active in the group do go into nursing homes and places like that and try to educate the caregivers as well as family members as to what this disease is.

Quite often, I receive a phone call at home with people, it is their first contact with someone who has a family member with Alzheimer's. They start telling me their story, and they are amazed to know that I have heard this story before.

Every family has a different story, but our similarities are great, and they are finding out for the first time that they are not in this alone, other people have the same problem, and that is the greatest support and comfort that we can give to people.

Mr. GORE. Of course, the medical profession, like all the rest of us, has become aware relatively recently how come the illness is, and all of the implications of the illness, but certainly this approach to patients has got to change.

I am confident there are people within the medical profession who are pushing for this change, but families need to be told and they need some help in mapping out their life strategies for dealing with this tragedy.

Mrs. BURNS. I do believe that the doctors are more alert about that now. I like to be kind and think our doctors really didn't know 10 years ago, or have the knowledge that they do now.

We have doctors on our board with our local Alzheimer's organization and they are very good. We go into hospitals and give talks to hospital personnel, and one of the greatest chores is doctors educating doctors, because they are very hesitant to change their diagnosis of hardening of the arteries to Alzheimer's.

Mr. GORE. Mrs. Grashot, Congressman Winn spoke earlier about nursing homes. Do you have any thoughts you would like to share with us about the comparison between nursing home care with respite care available?

Mrs. GRASHOT. A great deal. First of all, we have fought for some time for one little small thing in three nursing homes at home. They take to each room a pitcher of water and glasses. My people cannot reach for and get that glass of water, and yet, that is their fluid for 8, 10, 12 hours.

They are not educated as to the needs of an Alzheimer's patient, and a lot of reasons. They are understaffed, no doubt about that, but also, the education, I keep going back to the love and affection, if they were taught classes of this type, and the University of Tennessee in Memphis is doing an injustice to the nursing homes, just because they are all in the same city, they could send people out to educate these people in the gerontological department, and I am going to be in trouble, but anyway, they could do these things.

It is not that much. Go back to just love and attention. You can't give anybody something over here that is an Alzheimer's patient and expect them to be able to get it. I saw my husband in restraints, and not only the light weight, the belt, in heavy leather straps.

It is not the thing to do. At the time, you just really don't know whether it is, whether to say yes or no or how to cope with it, but my Louis did go through every stage of an Alzheimer's patient.

As my daughter said once in the latter stages, "We did miss the seizures mother," but while he was in the hospital, we had the seizures, so we went through everything there was to go through with an Alzheimer's patient.

I just don't feel mainly, talking about the nursing homes, if they are at that stage where they have to be strapped down, I don't think that is the thing to do.

If, prior to that, they are there and are mistreated, which they are, because of lack of knowledge, because of people looking for the paycheck rather than looking to the patient, they are tied not only to that, maybe not tied down with straps, but they are limited to this little room, which they only see that room. They don't get out.

I have a whole new concept of nursing homes, too. It, too, needs the big freedom, the big area, 2 or 3 acres of space for my people to roam. That is important, the freedom that they deserve.

We have it. We enjoy it, and they should, too.

Mr. GORE. We could ask a number of other questions of this panel, but we have two other large panels to go through today, and we are running over our time. I would like to thank all three of you for testifying here today.

It is important to have your experience, your knowledge and your statements here, because you are on the front lines.

You know more about it than most anyone else, and as we try to figure out the most appropriate response, including the research effort, to step it up, to find a cure for this disease, including better efforts to assist the primary caregivers such as members of the immediate family who are taking this responsibility.

As we undertake those efforts, we will certainly be using your statements and your experiences in that effort.

I want to thank you again for being with us today.

Mr. GORE. I would like to call our second panel of witnesses. Dr. Franklin Williams, Director, NIH National Institute on Aging; and Dr. Murray Goldstein, Director, National Institute of Neurological and Communicative Disorders and Stroke, National Institutes of Health; and Dr. Lawrence Silvers, Acting Director, National Institute of Mental Health, National Institutes of Health.

All of your statements will be entered in the record in full. This is a very pretigious panel, I might add, but I want to take special note of that fact.

Thank you for being here. Dr. Williams?

STATEMENTS OF DR. FRANKLIN WILLIAMS, DIRECTOR, NATIONAL INSTITUTE ON AGING, NATIONAL INSTITUTES OF HEALTH, BETHESDA, MD; DR. MURRAY GOLDSTEIN, DIRECTOR, NATIONAL INSTITUTE OF NEUROLOGICAL AND COMMUNICATIVE DISORDERS AND STROKE, NATIONAL INSTITUTES OF HEALTH, BETHESDA, MD; AND DR. LAWRENCE SILVERS, ACTING DIRECTOR, NATIONAL INSTITUTE OF MENTAL HEALTH, NATIONAL INSTITUTES OF HEALTH, BETHESDA, MD

Dr. WILLIAMS. Mr. Chairman, my colleagues and I certainly applaud the concern and interest of your committee in promoting a better understanding of Alzheimer's disease.

I simply want to hit a few of the highlights in my prepared statement, and I certainly will—all of us will welcome comments, and the questions, obviously, at the end of each of our statements.

In my own previous experience at the University of Rochester in New York, I worked very closely with colleagues in the many fields involved in research and teaching of Alzheimer's disease, and in the care of many patients with this condition and with their families, and now here at the National Institute on Aging, and in association with my colleagues in the other parts of the Public Health Service, I now have the opportunity to see even more clearly the extent and importance of this disease; and the students for Progress Through Research and Training.

I would like to concur with the committee on the magnitude and seriousness of this devastating disease as has already been well-noted in the earlier presentations.

Most of our knowledge of Alzheimer's disease has been acquired during the past 10 years due largely to the research supported by the Institutes represented in this scientific panel.

Less than 10 years ago, all but a handful of experts in the scientific and medical communities believed that Alzheimer's disease was a rare brain disorder which typically occurred in persons in the early 1950's, and, at the same time, it was generally accepted that forgetfulness, confusion, and other so-called senile-like symptoms were a normal part of aging.

That is what we heard from the persons who just spoke. Through the leadership of these three Institutes and scientists around the Nation, Alzheimer's disease has very rapidly now become recognized as an abnormal condition of the aging brain and not a natural consequence of aging.

During this decade, research accomplishments have been made at a number of levels. At the structural level, we now recognize clearly the characteristic changes of this disease and very characteristic areas of the brain, the tangled fibers normally called neurofibrillary tangles, and the plaques that left debris left behind by degenerating nerve cells.

A crucial finding has been the discovery that the tangles inside the Alzheimer's brain are made up of an unusually insoluble protein.

We also know that the nerve cells in the brains of healthy older persons show continued regrowth of dendrites, the branch-like extensions between nerve cells through which messages are received.

In Alzheimer's disease, this regrowth apparently does not occur. The dendrites either stop growing or degenerate.

At the neurochemical level, we know that there is marked loss or deficiency of acetylcholine and its synthesizing enzyme.

In metabolic studies, such as using the PET scanner that Senator Heinz referred to, it has been shown that brain metabolism is decreased with advancing disease.

This research progress continues apace, and almost every other issue of the magazine Science in the last several weeks has had an article on progress and research in Alzheimer's, and I know you will be hearing from some of the investigators who presented that research later in your hearings, so I won't recapitulate it here.

At the level of possible causes of Alzheimer's disease, there is much research activity. There are leads to suggest that Alzheimer's disease could be the result of a slow virus or virus-like agent. There are also other studies that implicate possible environmental toxins, and a third group of studies are focusing on the role of genetic predisposition, the role of genetic inheritance, at least some people with Alzheimer's disease.

At the more clinical level, there is increasing recognition of the absolute necessity to carry out a thorough differential diagnosis and to rule out potentially treatable causes of dementia before concluding that a person has Alzheimer's disease.

Too often multiple diseases in the elderly speed the progress of the disease, or obscure the symptoms of Alzheimer's disease. There is much work going on which attempts to define and understand better the manifestations of this disease, the precise nature of the cognitive, linguistic, and sensorimotor deficits, central motor changes, the variations in the course of the disease, and also the impact on the families of the affected persons.

At the level of treatment, research efforts are underway in a number of settings to test the effectiveness of various drugs on brain function. To date, these efforts are still at the experimental stage.

It is too soon to state whether they might be effective; but even without an effective drug therapy, we have learned much can be done to help the patients and their families live with this disease.

We have heard some vivid examples here. The most practical aids are carefully structured regular daily living schedules in family settings, good medical care, selective use of home support services, the use of day programs, and information and support of other types for the family members.

The help of such groups as the Alzheimer's Disease and Related Disorders Association have been very important for so many people with this condition.

We need to learn more about what types of respite care can be the best substitutes and help for these families.

Future priorities I want to summarize by saying that, first of all, in my judgment, we need to continue to pursue most vigorously the promising leads that we have in research, both in the basic mechanisms and possible causes of the disease, and also in the area of defining more clearly the clinical picture and manifestations of this disease.

We certainly need to continue to work to improve our diagnostic and treatment skills.

Another need is to be more effective in transferring the most up-to-date information to the practicing professionals who are providing care, and your questions, Mr. Chairman, with the recent witness just bear that out, how much the whole range of practicing professionals need to be better informed in order to give more proper care.

A major barrier to progress in my judgment is the scarcity of trained leaders, teachers and investigators, in all of the relevant disciplines.

As has already been mentioned this morning, there is a tremendous need for well-prepared persons to teach medical students and those of the other professions, to provide continuing education for the already-practicing professionals, and consultative services and most importantly, to carry on the many avenues of promising research.

Some promising steps are being taken with the task force set up by the Secretary of Health and Human Services, whose report is imminently to be issued, and all of us were active members of that task force.

The appropriations by Congress of $3.5 million in this fiscal year for the National Institute on Aging, in collaboration with the other Institutes represented here, to establish Alzheimer's Disease Research Centers and the prospect that we may be able to extend those numbers next year.

In this year, we received 22 applications for what we could have funds, for up to five such centers, and the majority of those applications were very high quality. It is clear that the science community is able and ready to proceed much more rapidly in addressing the problems in this disease in multidisciplinary research and training efforts.

I would just close by saying that I believe we have made really remarkable progress in a very short time in understanding Alzheimer's disease, and that we will see much further progress in the next few years.

We must be cautious in our estimates of how soon or how far we may be able to go in correcting the devastating effects of this disease, and until there is a major medical breakthrough, however, we can certainly offer patients and their families the hope that we can lighten their burden through good medical care, community services that can help meet their everyday needs and increase public understanding.

[The prepared statement of Dr. Williams follows:]

STATEMENT BY

T. FRANKLIN WILLIAMS, M.D.

DIRECTOR, NATIONAL INSTITUTE ON AGING

NATIONAL INSTITUTES OF HEALTH

I applaud the concern and interest of your committee in promoting a better understanding of Alzheimer's disease. I see the problem of Alzheimer's disease from the perspective of an educator, a physician, an administrator, and a researcher. Before coming to the National Institute on Aging (NIA), I served as co-director of the University of Rochester Center on Aging and head of geriatric programs at Monroe Community Hospital and, in that capacity, I worked closely with colleagues in the many fields involved in research on Alzheimer's and in the care of many patients with this condition, and with their families. Here at the NIA and in association with my colleagues in other parts of the Public Health Service, I now have the opportunity to see even more clearly the extent and importance of this disease and the opportunities for progress through research and training.

Let me first emphasize the magnitude and seriousness of this devastating disease. Although, fortunately, the great majority of people maintain good mental functioning into their later years, the proportions of those who acquire a significant degree of dementia rise rapidly among the very old. Overall, current figures suggest that only 5 to 10 percent of the 27 million Americans over age 65 suffer the cognitive deficits formerly thought to be a normal part of old age. But between the ages of 60 and 80, the risk of developing dementia doubles every 5 or 6 years. Current projections indicate one third of the entire population in developed countries will live to age

80 and beyond when there is an estimated one in five chance that a person will have serious, irreversible dementia. Furthermore, the over 85 age group is the fastest growing segment of the older population in the United States. By the year 2000, only 15 years away, there will be over 5 million Americans aged 85 and older. Our best efforts are needed to minimize disease and help maintain function and independent living for all of us.

I would like to address three important aspects of this problem: 1) current status of knowledge about Alzheimer's disease; 2) priorities for further progress; and 3) hopes and opportunities for the future.

Most of our knowledge of Alzheimer's disease has been acquired during the past 10 years, due largely to the research supported by the Institutes represented on this scientific panel. Less than 10 years ago, all but a handful of experts in the scientific and medical communities believed that Alzheimer's disease was a rare brain disorder which typically occurred in persons in their early 50's. At the same time, it was generally accepted that forgetfulness, confusion, and other "senile-like" symptoms were a normal part of aging. In 1977 and 1978, two major international conferences were sponsored by the National Institute of Neurological and Communicative Disorders and Stroke, the National Institute on Aging, and the National Institute of Mental Health. These conferences focused on the pathological and clinical relationship betwen senile brain disease and

Alzheimer's disease. In 1979, an NIH consensus development conference on treatment possibilities for mental impairment in the elderly helped focus attention on the difference between normal aging and dementia.

As a result of these and other efforts, Alzheimer's disease is today recognized as the most common cause of severe intellectual impairment in older individuals. Whether the presenile form of Alzheimer's disease is identical in cause, mechanisms, and course to Alzheimer's disease in the elderly is still not completely established. Nonetheless, Alzheimer's disease is recognized as an abnormal condition of the aging brain and not a natural consequence of aging. This is enough to encourage medical science to study the disease and develop a foundation for effective treatment and prevention.

During the last decade, research accomplishments have been made at a number of levels:

At the anatomical, structural level, we now recognize the characteristic changes of this disease. By examining brain tissue of persons who have died with the disease, we see masses of tangled fibers called neurofibrillary tangles within certain nerve cells, as well as placques, the debris left behind by degenerating nerve cells. These changes can to a degree be correlated with the severity of the changes in memory and other brain functions that a patient experiences. A crucial finding has been the discovery that the tangles inside the Alzheimer brain are made up of an unusually insoluble protein. Furthermore, the enzyme transglutaminase has been linked to the formation of this abnormal protein. This is an important clue about how these proteins develop.

We also know that the nerve cells in the brains of healthy older persons show continued regrowth of dendrites, the branch-like extensions between nerve cells through which messages are received. In Alzheimer's disease, this regrowth apparently does not occur. The dendrites either stop growing or degenerate.

At the neurochemical level, we know that there is marked loss or deficiency of acetylcholine and its synthesizing enzyme. Acetylcholine is one of several neurotransmitters, chemical compounds produced within nerve cells to allow communication between cells. This deficiency occurs in the areas of the brain that are responsible for learning and memory. There may also be changes in other neurotransmitters.

At the metabolic level, there is evidence that changes in brain metabolism affect the production of acetylcholine. In the NIA Laboratory of Neurosciences at the Warren G. Magnuson Clinical Center, scientists are using the PET (positron emission tomographic) scanner, the most advanced approach to measure and localize metabolic activity in the brain. These studies are showing that the Alzheimer brain uses less glucose, the main source of brain energy and the substance necessary for the production of acetylcholine. There is also evidence of decreased amounts of one of the key enzymes, pyruvate dehydrogenase, needed for breaking down glucose and synthesizing acetylcholine.

The research progress continues: Just within the past month, there has appeared an important report of findings on the apparent mechanism by which the synthesis of the functional proteins in brain cells is decreased in Alzheimer's disease. In diseased autopsied brains, scientists at Harvard Medical School found little more than half the normal amounts of RNA and an excess of an enzyme that normally breaks down RNA.

At the level of possible causes of damage to the brain in Alzheimer's disease, there is a great deal of research activity. One promising lead suggests that Alzheimer's disease could be due to slow virus or virus-like agent. Other forms of dementia such as kuru and Creutzfeldt-Jakob diseases have been shown to be caused by these agents. Recent research suggests a similarity between the amyloid deposits in the brain tissue of Alzheimer victims and virus-like particles found in a dementing disease in sheep. A second lead supports the possibility that Alzheimer's disease is due to an environmental toxin such as aluminum which could affect brain cell metabolism. A third group of studies is focusing on the possible role of genetic predisposition.

At the clinical level, there is increasing recognition of the absolute necessity to carry out a thorough differential diagnosis and to rule out potentially treatable causes of dementia before concluding that a person has Alzheimer's disease. Too often, multiple diseases in the elderly obscure the symptoms of Alzheimer's disease, or speed the progress of the disease. In the Fall of 1983, the NINCDS, with collaboration from the NIA and NIMH, convened a workgroup of clinical investigators who prepared a guide to diagnosis of Alzheimer's disease which was published in the journal Neurology and which has now been widely circulated. In addition, in December 1983, the American Association for Retired Persons, in collaboration with NIA, NINCDS, and NIMH, jointly sponsored a research workshop on the diagnosis of Alzheimer's disease to identify areas of scientific opportunity and develop future plans for research on diagnosis.

There is much work going on which attempts to define and understand better the manifestations of Alzheimer's disease; the precise nature of the cognitive, linguistic, and sensorimotor deficits associated with this disease; the variations in the course of the disease; and the impact on the families of affected persons. Two important goals are to learn enough about the disease to develop more sensitive and reliable diagnostic tests and to improve patient care.

At the level of treatment, efforts are under way in a number of research settings to test the effectiveness of various drugs on brain function. To date, most of these studies have attempted to increase or maintain the levels of acetylcholine in the brain and to test other substances for their ability to improve the responsivity of the traget nerve cells to the action of acetylcholine. These efforts are still at the experimental stage; it is too soon to state with any confidence whether they might be effective.

But even without an effective drug tnerapy for Alzheimer's disease, we have learned that much can be done to help patients and their families manage and live with this disease. The most practical aids are carefully structured, regular daily living schedules in familiar settings; good medical care; selective use of home support services; and information and social support for the family members. With the help of such groups as the Alzheimer's Disease and Related Disorders Association, most families get along remarkably well for long periods of time, but at what is often a heavy emotional and physical price for everyone. Arrangements for respite for the family members through temporary substitute care for the affected person are most important.

Even if Alzheimer's disease is present, it is important to help the affected person remain independent for as long as possible. Emphasis on managing the multiple problems facing elderly persons, and on maintaining health and effective functioning, are constant concerns of the NIA. In keeping with these goals, the NIA in 1983 conducted a very well-attended state-of-the-art conference on functional assessment methodology. This meeting was co-sponsored by the American Medical Association, the National Center for Health Services Research, and NIH's Office of Medical Application of Research.

What are the priorities for further progress?

In my view we need, first, to continue to pursue vigorously the promising leads in research, on the basic mechanisms and possible causes of Alzheimer's disease, such as those that I have referred to.

Second, we need to have a clearer picture of the clinical manifestations of Alzheimer's disease. Before we can hope to make further progress, we need to know more about the disease's earliest signs and symptoms, the changes it causes in behavior, its overall course, and its prevalence. Because serious study of the disease only began 5 to 10 years ago, and also because firm diagnosis has been possible only at autopsy, we have very limited knowledge about the full spectrum of the disease. Up to now, our experience has been largely limited to patients in advanced stages, possibly too late to respond to therapy.

Third, we need to achieve more interdisciplinary teamwork in both the medical and scientific communities in order to expand our knowledge and improve diagnosis and treatment of this condition in the patients and the families it affects. All of the levels of knowledge related to this condition referred to above interact with each other.

Fourth, we need to be more effective in transferring the most up-to-date information to the practicing professionals who are providing care. Too often, families are told that a relative has "cerebral arteriosclerosis"--a now outmoded concept--or that nothing can be done to help. Fortunately, confused older patients are given thorough diagnostic examinations more often than before, but even now, when the diagnosis is dementia, the family is rarely given help in planning daily routines, managing changes in behavior and personality, or using community resources.

Fifth, a barrier of major concern to me is the scarcity of trained leaders--including both teachers and investigators--in geriatric medicine and other relevant disciplines as they relate to care of the elderly such as neurology, psychiatry, psychology, pharmacology, nursing, or social work. There is a tremendous need for well-prepared persons to teach medical students and those of other health and social service professions, to provide continuing education and consultative services to practicing professionals, and to carry on the many avenues of promising research.

A promising step in addressing these priorities came when the Secretary established a task force addressing the need for coordinating research already being done in various agencies of the Department, to act as a mechanism by which all Departmental programs designed to serve the elderly can share information about the disease, to identify the most promising research leads, and to act as a vehicle to translate research into appropriate policies and programs. In addition, the Congress appropriated $3.5 million for FY 1984 to the National Institute on Aging to establish up to five Alzheimer Disease Research Centers. We have received 22 applications for such multifaceted centers and will very soon be announcing the awards of the first center grants in this field. The high quality of the majority of the applications is indicative of the fact that many research scientists are now prepared to move quickly to address all aspects of Alzheimer's disease. These centers are aimed at multidisciplinary research and training and have mechanisms built in to assure information transfer to the community.

What about hopes and opportunities for the future?

I believe what I have summarized documents the remarkable progress we have made in understanding Alzheimer's disease in a very few years. I believe that we will see much further progress in understanding the basic pathology and pathophysiology of Alzheimer's disease. I believe that before too long we will identify the cause or causes of Alzheimer's disease and thus be able to take steps to prevent it or halt its progress. But we must be cautious in our estimates of how soon or how far we may be able to go in correcting the devastating effects of Alzheimer's disease through medications. Until there is a major medical breakthrough, we can offer patients and their families the hope that we can lighten their burden through good medical care, community services that can help meet their everyday needs, and increased public understanding of their plight.

Thank you, Mr. Chairman.

Mr. Gore. Thank you very much.

We will hold off on questions until the end of the panel. Dr. Murray Goldstein, Director, National Institute of Neurological and Communicative Disorders and Stroke, National Institutes of Health. We are honored you are here. Please proceed.

Dr. Goldstein. Thank you for the privilege of testifying before you. The written statements of the three Directors in charge of the three Institutes summarize quite well the state of the art in terms of accomplishments and research opportunities, and so, with your permission, I won't repeat those, but let me try to highlight a few issues which you might find helpful.

First of all, there has been some breakthroughs and we must recognize that.

The most important breakthrough on Alzheimer's disease, previously called senile dementia, is to recognize that it is a disease, that it is not a normal consequence of just getting older. That is not true.

It is a disease. It is like other diseases, and hopefully through research, we will be able to prevent it, and where we fail to prevent it, treat it.

The second important issue was that for too many years we thought of it as a general degeneration of the total brain.

What we were looking at was the consequence of the disease and not the disease. Some very able scientists later today will testify to you about their own findings, which have demonstrated that it is probably a focal disease, a disease which starts in one area of the brain and then begins to affect the other areas.

This is very important, sir, because now perhaps we know where to look and not be confused by the general pattern, but be able to know where to look, so, hopefully, we can intervene.

The third is that we are beginning to understand a whole new world of brain diseases, and these are diseases that are biochemical in origin, and in modern terms, we call these neurotransmitters, the materials which one cell sends to another cell, and that is how they talk to each other, and when they fail to talk, then we have a kind of disorder we are discussing, and so, these three accomplishments over the last 10 years have really been amazing.

I think I would like to compliment the scientific community for them, because on those three firm accomplishments, we now have the opportunity to begin to look intelligently at what we are going through.

A second important issue, sir, is that I would like to compliment the Congress, because it is now turning its attention to the next big problem of medical research.

For many, many years, and quite properly, we focused our attention on the great killers of our society, heart disease, cancer, stroke, and the last, of course, something we in the Neurological Institute are very concerned about, but now, the Congress is beginning to turn its attention to the great debilitators as well.

The diseases that affect our quality of life, which makes life worth living. In some respects, these are more difficult problems, but as one witness previously said, it is the issue of the living dead, the issue of people who may be alive, but whose lives are in a dreadful state, and Alzheimer's disease is a wonderful example of that type of attention, and I would hope not only are we discussing Alzheimer's disease, but debilitating diseases in general.

Let me elaborate on one very exciting development Mr. Carney touched on, and Senator Heinz touched on it, and that is the whole issue of brain imaging. Up until very recently, we were not able to look inside the human brain.

Yes, we did have x ray, but x ray tells us only anatomy. It tells us what the structures look like, which is very important in many, many illnesses.

CAT scanning is really another way of doing x rays, more sophisticated, using computers, having finer technology, again very important.

Sir, if we had a person who died and within 2 minutes took an x ray or a CAT scan of their brain, their brain would look perfectly normal. What they would be telling us is anatomy.

Today, we can watch the human brain think, and we can watch it work, and that is called PET scanning, positron emission tomography. We can feed a person sugar which has a label on it and through the use of a PET scan, which doesn't injure the person in any way, we can ask the person to do tasks.

As an example, we could ask a person, let me see you think. Don't do it. Just think about moving your thumb, and part of the brain lights up, and as this part of the brain begins to increase its consumption of fuel, sugar, glucose, we can then ask that person to begin to move that thumb and watch the other parts of the brain become incorporated into that action.

That is what we mean by PET scanning. There is a PET scanner at Brookhaven working with a group at Stony Brook at New York University, so we can now begin to look at disorders of the function of the brain and the diseases of function of the brain.

Where this will take us, I don't know yet. It is really quite new, and we are moving along vigorously with that, but this is just one of the technological breakthroughs which have given us the opportunity to ask the intelligent questions.

It wasn't that we were not doing things before, but we did not have the opportunity because of the methodologies unavailable to us, and they are becoming available.

Finally, it has been very true that the physicians of this country did not know enough about Alzheimer's disease. They did not recognize it. They didn't understand it.

It was a mysterious rarity and, therefore, the advice given to patients is too sparse. Second, the misdiagnosis, 20 percent were misdiagnosed. People who did not have it were being called Alzheimer's. The other 20 percent were perhaps treatable.

The task force took the development of information for the physician, and I am very delighted to say that under the auspices of the task force, this material is now being spread nationwide, and it is a brochure written in terms that all physicians at every level of practice can and shall understand, called "Alzheimer's Disease, a Scientific Guide to Help Practitioners," and I brought some copies for the committee.

Under the auspices of the task force, we have been able to develop the information which will now permit the physician to understand the disease himself and to communicate adequately with his or her patient, so we are moving on this, sir.

Hopefully, we can accelerate as we learn more and can do more, but we are moving ahead with it.

Thank you for the opportunity to testify.

[The prepared statement of Dr. Goldstein follows:]

ALZHEIMER'S DISEASE - TESTIMONY

Dr. Murray Goldstein, Director
National Institute of Neurological and Communicative Disorders and Stroke

Mr. Chairman, I am the Director of the National Institute of Neurological and Communicative Disorders and Stroke (NINCDS) of the National Institutes of Health (NIH). The NINCDS conducts and supports research on the neurological disorders of the brain and spinal cord and those uniquely human characteristics of speech and language. This includes research on Alzheimer's disease and on other neurological disorders which share clinical features of dementia, such as Parkinson's disease and Huntington's disease. I am here today to summarize for you examples of that research, specifically, what neurological research has taught us about Alzheimer's disease. I am going to discuss signs and symptoms of Alzheimer's disease as well as the several different hypotheses of its causes. Finally I will provide examples of ongoing neurological research on Alzheimer's disease.

Diagnosing the Alzheimer's Disease Patient

Before I summarize what is known about the basic disease process, I want to clarify that I am using the term Alzheimer's disease to include all dementias of the Alzheimer's type, regardless of the age of the patient. I am discussing a dementia that is due to specific pathological changes in the brain. Thus, we include both those persons in their 40's, 50's and early 60's who have what was called Alzheimer's disease in the past, as well as those older Americans with dementias of the Alzheimer's type. We wish to differentiate patients with Alzheimer's disease from those with depression or drug-induced problems and from those with multi-infarct dementia caused by "hardening of the brain arteries" or by multiple small strokes.

The Alzheimer's disease patient is brought to the physician because the family has recognized startling changes in mental abilities, personality and behavior or marked confusion, disorientation and memory loss. The forgetfulness of Alzheimer's disease is far different than the transitory forgetfulness or absentmindedness experienced at any age. Unlike the benign forgetfulness of not remembering where you put your glasses, the malignant forgetfulness of Alzheimer's disease means that the patient forgets that he or she wears glasses at all. While benign forgetfulness is general and sporadic, the forgetfulness of Alzheimer's disease is very specific. Patients may no longer remember how to dress properly, for example, how to tie their shoelaces or close a zipper, and keep making the same mistake each time they dress. The patient often may show signs of hostility or evasiveness even to loved ones who are the closest family members.

When confronted with a patient whose dementia may be due to Alzheimer's disease, physicians conduct a range of neurological tests to determine if the patient's confusion and other symptoms have a neurological basis in the organic structure and function of the brain. Patients with Alzheimer's disease will demonstrate this organic basis to their disease. The patient's reflexes will be examined, as will his or her stance or walk. Like Parkinson's disease, Alzheimer's disease has particular movement signatures. Many Alzheimer's disease patients have a slow gait with drooped posture. Often, arm swing is almost absent and they take mincing steps from a wide-based, flexed stance.

To test the patient's higher functions, the physician will use an oral test, called the Mental Status Questionnaire (MSQ). This test is comprised of 10 simple questions, such as "Where are we now?" "What is today's date?" "What month is it?" "How old are you?" "When is your birthday?" "Who is the President of the United States?" and, "Who was the President before him?" While patients in the early stages of Alzheimer's disease may score reasonably well on this test, many Alzheimer's disease patients will rate as severely or moderately confused.

The patient's ability to travel alone on public transportation, to get dressed without help, and to bathe without assistance are also considered as is the ability to shop, cook, or do ordinary household tasks alone. Tests of this sort specify the patient's level of impairment.

At the first visit to the physician, definitive diagnosis can be difficult. Other neurological or psychiatric disorders can present with features that resemble the signs and symptoms of Alzheimer's disease. A major characteristic of Alzheimer's disease is its continuing downhill progression. Physicians suspecting the disease in a patient will repeat the initial round of tests at six-month intervals. If the patient has worsened, Alzheimer's disease may well be the diagnosis.

Because diagnosis of Alzheimer's disease is so difficult and often inconclusive, the PHS recently issued two publications designed to increase health professionals' knowledge of the disease. One, "Alzheimer's Disease: A Scientific Guide for Health Practitioners," discusses the disease, what we understand of its etiology, and the office tests physicians may use to determine a diagnosis when a patient presents with symptoms suggestive of Alzheimer's disease. This booklet differentiates Alzheimer's disease from a multitude of other causes of mental impairment, some of which are reversible and can be treated. The Institute also published reprints of a July 1984 article in the journal Neurology entitled "Clinical Diagnosis of Alzheimer's Disease." This paper was prepared by a work group of scientists and physicians under the auspices of the Department of Health and Human Services Task Force on Alzheimer's Disease. It defines specific criteria for clinical diagnosis of the disease and specifies three different diagnosis categories: probable, possible and definite Alzheimer's disease, as well as criteria for classification of the disease for research purposes. Aspects of medical history, clinical examination, neuropsychological testing and laboratory assessments that can contribute to a diagnosis of probable Alzheimer's disease are described in the article. These publications are being widely disseminated to health professionals and we believe they will aid individual physicians and improve the accuracy of diagnosis.

Basic Theories of Alzheimer's Disease

At this time the definite diagnosis of Alzheimer's disease can only be made under the microscope, usually at autopsy. The brain of the Alzheimer's disease patient has several unique characteristics. It has specific anatomical lesions called neurofibrillary tangles and neuritic plaques. Neurofibrillary tangles are made up of fine nerve filaments twisted around each other. They are found in the cell bodies of the brain cells.

Research has disclosed that the tangles are made up of an abnormal protein that is particularly insoluble and hard. The implication of the insolubility of the tangles is unclear. Yet, we are confident that the tangles should not be in the brain and are probably there permanently.

The neuritic plaques are also composed of an abnormal protein. We believe them to be degenerating bits of the nerve cell endings that surround a core of fibrous protein material. They are sometimes called the senile plaques. The protein in the core is similar to the protein which makes up antibodies. I have elaborated on the plaques and tangles in the brain because they are crucial to our ideas of the cause of Alzheimer's disease. We do not yet understand them, but we have observed that they are associated with the severity of the patient's disease: the more plaques and tangles there are, the more severe the patient's symptoms. The number of tangles and plaques is an indicator of the progression of this organic disease of the brain. We do not know if they are a result of the disease or, perhaps, a contributing factor.

In the mid 1970's three independent groups of scientists, all working in the British Isles, discovered that a particular enzyme, choline acetyltransferase--CAT for short, was practically gone in the brains of patients dying with Alzheimer's disease. CAT is involved in the process by which a key neurotransmitter, acetylcholine, is made in the brain cell. Other scientists had demonstrated earlier that acetylcholine was the chemical used by nerve cells to communicate with each other and is probably involved in memory.

We have recently learned the probable location in the brain of the changes that are involved in the memory loss and general deterioration of Alzheimer's disease. Scientists at The Johns Hopkins University discovered that cells deep in an area underlying the mantle of the brain, the basal forebrain, are the site of the lesions I've just described. The nerve cells in this part of the brain send fibers to many parts of the mantle of the brain and to a specialized area of the brain important to memory called the hippocampus. These nerve cells use acetylcholine as their neurotransmitter. In Alzheimer's disease patients, the Johns Hopkins' scientists discovered, the basal forebrain neurons had degenerated. We believe that the death of these cells may cause the brain damage of Alzheimer's disease, leading to the clinical symptoms of intellectual loss. Thus we can now tie together the loss of the enzyme CAT with the specific location of the neurons which die in Alzheimer's disease.

We do not yet know why these neurons die or what causes the neurofibrillary tangles and senile plaques. There are several hypotheses offered to explain the situation--specifically, to explain what causes Alzheimer's disease. In one approach, scientists are testing the idea that the body mounts a self destructive attack as a response to the abnormal proteins seen in the tangle and the plaque. On the other hand, we must recognize that the tangles and the plaques may be the result of the brain's attempt to protect itself against some as yet unknown invader.

The blood-vessel theory is another possible explanation of the disease. The blood-brain barrier is a cellular wall that protects the brains of healthy people from materials that could enter the brain from the blood stream; viruses, bacteria, and other substances that could possibly harm the brain. According to this theory, the blood-brain barrier is violated and the culprit enters the brain substance and damages it in such a way as to cause Alzheimer's disease. However, the nature of the culprit or an explanation of why the blood-brain barrier is open remains to be explained.

The immunological and blood-vessel theories provide alternative explanations of how the brain is devastated. But what affects the brain? What activates the immune system or passes through the blood-brain barrier? Scientists have several different theories. One is the genetic theory. Although Alzheimer's disease doesn't appear to follow strict Mendelian laws of inheritance, the disease sometimes occurs in families. While the average person's chance of developing the disease is about 1 percent, the odds increase four-fold or more if a close relative is affected. There is no clear-cut genetic component but we suspect a genetic aspect, perhaps a genetic predisposition or propensity to the disease. We are now studying those families in which there are clusters of victims.

Another theory of the causation of the disease is the viral theory. Scientists have noted that patients with kuru, another dementing disease, have similar brain plaques. Kuru is a viral disease of the brain caused by one of a class of agents known as slow viruses. These organisms incubate in the nervous system for decades before becoming active and causing damage. Investigators have also seen similar plaques when they inoculate mice and hamsters with scrapie, another slow virus that ordinarily affects sheep. Important in its own right for its significant damage to sheep, scrapie provides a potentially valuable animal model and allows us to investigate this viral theory.

The environmental theory offers still another explanation. Aluminum, the third most common element, is found in higher than usual concentrations in the brains of Alzheimer's disease patients. Aluminum has been found in the neurofibrillary tangles by scientists in our Intramural Research Program at Bethesda. Aluminum, as we know, is toxic to the brain and causes abnormal brain function. Research on the role of aluminum and other minerals is continuing.

Research Leads

The PHS is currently conducting a full slate of research on Alzheimer's disease. The Positron Emission Tomography (PET) Program is evaluating human brain metabolism in various disease states, including Alzheimer's disease. The basic research the PHS supports and conducts should uncover clues into the cause, treatment, and eventually prevention of Alzheimer's disease. Research on the organization of the brain and its neurotransmitters will uncover new information that promises to lead to a better understanding of the brain, the way we think, and how our memory works. Eventually this work should help us understand Alzheimer's disease.

Specific research is also targeted on the disease process. Time does not allow me to catalog that work in detail, but a few examples may be useful. Scientists funded by the Public Health Service are studying a putative new agent, the prion, to determine if Alzheimer's disease has an infectious origin or component. Scientists, also supported by PHS funds, are using immunological techniques to study the origin of neurofibrillary tangles and other facets of the disease. Investigators are studying the unusual properties of the tangle proteins which make them so resistant to solution, and one group of scientists is tracing the specific connections of the nerve pathways involved in higher mental functions. Hormones and neurotransmitter relationships in the brain are being studied by scientists with Public Health Service support.

Applied research is also under way. Positron emission tomography (PET) scans of Alzheimer's patients have allowed us to correlate specific metabolic changes in the brain with specific symptoms in Alzheimer's disease. We can see that energy utilization is greatly reduced in a specific part of the brain that we know is associated with the ability to solve simple puzzles in those patients who have lost that particular skill. In patients whose ability to use the appropriate words in conversation is impaired, the areas of the brain involved in speech have greatly decreased function. The Public Health Service is funding scientists who are trying to devise more accurate tests for early diagnosis of Alzheimer's disease. These investigators are conducting a longitudinal study of an aging population and hope to increase our understanding of the natural history of Alzheimer's disease and other progressive dementias. From studies like this, we should get better ideas of risk factors for the disease.

In summary, we now can say with certainty that Alzheimer's disease is a disease of the brain--it is not a normal consequence of the aging process. It is a disease--and therefore, should be preventable and treatable. Scientists on the NIH campus and in laboratories around the country are working towards this objective. The NINCDS is actively participating in the Secretary's Task Force, which I am certain will be most helpful in coordinating these scientific efforts and bringing their results rapidly into clinical practice.

Thank you for the opportunity of describing these exciting scientific advances being made about this devastating disease. I can assure you every research arm of the Public Health Service is actively involved in solving the riddle of Alzheimer's disease. I also assure you that there is hope through research--hope for prevention and improved therapy. I would be pleased to answer any questions the Subcommittee may have.

Mr. GORE. Dr. Lawrence Silvers, Acting Director, National Institute of Mental Health, National Institutes of Health. Please proceed.

Dr. SILVERS. I am delighted to be here. The National Institute of Mental Health works closely on and collegially with the National Institutes of Health, but we are part of a separate research agency, the Alcohol, Drug Abuse, and Mental Health Administration.

Mr. Carney and others talked about raising the research budget for NIH, and I need to remind Congress that there are two research agencies within the Public Health Service.

Research on Alzheimer's disease is one of the highest priorities for Secretary Heckler, and she put together a departmental task force almost immediately upon coming into her office; and by working with Assistant Secretary Dr. Brandt, who chaired this task force, facilitated the completion of its work.

This report will be coming out shortly, but it will highlight the need to coordinate intense research within the Public Health Service, to pinpoint promising areas of future research, to act as the vector translating these research findings into policy, programs, and practical means for improving the quality of life for Americans, to develop an active ongoing mechanism for sharing information with other program heads in the Federal agencies which serve the elderly, such as the Health Care Finance Administration and Administration on Aging.

I would like to focus on some of this report and especially on the part that NIMH is proud to have taken part in handling. I will not repeat some of the research issues that are being discussed by the other Institutes and that will be in the formal reports, but would like instead to focus on the unique areas that NIMH has tried to work with the other Institutes.

And first is the area of clinical diagnosis. As you heard from the families earlier, and the other two Institute directors, this has been a difficult problem, and part of the confusion related to psychiatric diagnoses, specifically depression in the elderly that can stimulate dementia, and also, elderly who are on certain medications where the dosage may be too high for their age.

That may begin to show what in the past might have been called senile dementia, and the NIMH has worked hard to come up with a more accurate method for diagnosis and sort out those other disorders which are other mental illnesses.

While we work toward an understanding of the cause and effect of treatment, we are painfully aware of the present plight of the Alzheimer's patients, a disorder which includes both the patient and the family.

The significant contribution of the family to the Alzheimer's patient has been studied by some of our projects. We have been very active with the Alzheimer's and related disorder organizations.

It has also been documented severe stress and enormous burden, both financial and emotional, on such families, and we have funds at the University of Washington and Texas Research Institute of Medical Science in Houston to better understand this and better come up with models of assistance and intervention.

So often, one finds a person with Alzheimer's disease and a related close family member who develops depression, as we heard from one family member this morning, and we are trying to be available to help in this area as well.

It is clear that research on Alzheimer's disease requires a multidisciplinary, multifaceted approach. We need to continue vigorously to expand our research for the cause of the disease. We need better data on its epidemiology.

We need specific diagnostic tests for both earlier and more accurate diagnosis of Alzheimer's disease, and more effective drugs and behavioral intervention treatments and to increase our understanding of how tó help the families.

We are strengthening our initiatives in each of these important research areas in a coordinated effort with other Federal programs, and especially with the other Public Health Service Institutes represented here today.

In an effort to meet these needs and to increase our knowledge base, NIMH has developed a new program of support, Clinical Research Centers on the Psychopathology of the Elderly.

These will be working very closely with the National Institute on Aging Centers relating to similar issues. This program weeds together clinical and basic research into the mental disorder.

Stanford University is attempting to identify areas of excess disability. Patients may be alleviated of these and begin to function better or have a better quality of life.

A second center on Alzheimer's disease located at the University of Washington in Seattle is actively developing refinements in the diagnosis and staging of the disorder.

We have a great concern for getting the necessary information out and have published a number of documents relating to the aging, which I will leave for the committee, and are pleased that we worked in getting the other material together for the primary care physicians who take care of these individuals.

In closing, I would also like to emphasize the collegial and collaborative relationships we have had with the other national research institutes.

Currently, research grant applications can be and are assigned jointly to as many institutes as appropriate so that each may consider the project for funding.

I thank you for allowing me to testify today, and I am extremely pleased that you are focusing on such a debilitating disorder.

[The prepared statement of Dr. Silvers follows:]

TESTIMONY

LARRY B. SILVER, M.D.

ACTING DIRECTOR, NATIONAL INSTITUTE OF MENTAL HEALTH

ALCOHOL, DRUG ABUSE AND MENTAL HEALTH ADMINISTRATION

I appreciate the opportunity to testify before this hearing of the Subcommittee on Investigations and Oversight of the Committee on Science and Technology. Research on Alzheimer's Disease is one of the highest priorities of Secretary Heckler. Upon taking office, she rapidly established a Departmental Task Force on Alzheimer's Disease and asked Assistant Secretary for Health, Dr. Edward N. Brandt, Jr., to chair the Task Force. Over the course of the past year, under the able leadership of Dr. Brandt, the Task Force has taken active steps to:

- Coordinate and speed the intensive research already being done at HHS on Alzheimer's;
- Pinpoint the most promising avenues for future research;
- Act as the vehicle to translate research findings into policy, programs and practical means for improving the quality of life for older Americans;
- Become an activist, ongoing mechanism for sharing information with other program heads in the Federal agencies which serve the elderly, such as HCFA and the Administration on Aging.

The Report of the Secretary's Task Force on Alzheimer's Disease will soon be issued. The report presents a detailed plan for research, training and services goals for the future, and includes an outstanding summary of current scientific activities. Copies of the report will be sent to the Committee for the record.

The National Institute of Mental Health's (NIMH) basic and clinical research efforts on Alzheimer's Disease are carried out through both research in our own intramural laboratories and support the research of investigators in the field through grants and contracts. Other activities at NIMH involve support for the education of researchers and clinicians, the development of statistical and informational materials, and national leadership in consultation with both public and private mental health service providers.

The focus for NIMH activities on Alzhiemer's Disease is the Center for Studies of the Mental Health of the Aging, one of eleven operating units of the Institute. The Center, made fully operational in 1976, has established Alzheimer's Disease as a major program priority. In 1976, the Center, in collaboration with the National Institute on Aging and the National Innstitute of Neurological and Commmunicative Disorders and Stroke, developed two research conferences on Alzheimer's Disease; proceedings of both have been published and are now considered standard resources in the field.

The conferences were the beginning of the focus on Alzheimer's Disease and also laid the framework for a series of collaborative efforts between the Institutes. Such collaborative efforts have involved the joint support of research grants and contracts, joint support of components of research programs at universities and medical centers, and the inclusion of cognitive and behavioral assessments in prevention-oriented clinical trials. Under interagency agreements the NIMH has also developed other joint program activities with the Administration on Aging, the National Heart, Lung, and Blood Institute, and the National Institute of Handicapped Reseach of the Department of Education.

The NIMH research in Alzheimer's Disease is, by design, multidisciplinary in the broadest sense. Investigators from the fields of neurochemistry to social work and from behavioral genetics to health care economics have joined with basic, clinical, and applied researchers in the range of biomedical, behavioral, and social disciplines that are involved in mental health research.

Exciting new findings are being reported and creative investigations are being pursued. This statement report conveys some of the excitement of research in this area.

With the increases in the absolute number and relative proportion of older persons in the general population, growing attention has been focused by NIMH on the devastating and disabling effects of those brain disorders which are associated with advancing chronological age. Among these disorders, Alzheimer's Disease has been estimated to affect more elderly persons than any other brain disease.

Until recently, this major disease was largely neglected by both laymen and physicians alike, and usually not distinguished from aging in general. However, in recent years, both have shown increased interest in this disorder. In the past decade a considerable amount of progress has been made in the understanding of Alzheimer's Disease by scientists around the world. Recent advances have radically altered our thinking about Alzheimer's Disease; whereas in the past, it was considered a normal consequence of aging, it is now known to be a disease entity.

In view of the great need for more fundamental information about Alzheimer's and the dementing disorders of middle and late life, we have actively embarked on a program of support to stimulate researchers to study the problems. The Institute currently supports a wide-ranging multidisciplinary set of studies which have both theoretical and applied implications: these include studies of the epidemiology, etiology, diagnosis, natural history, management, and treatment of Alzheimer's and related brain disorders. The Institute's interests range from support of studies applying advances in positron emission tomography (PET) and problems in differential diagnosis, to systematic investigations of the role of the family in sustaining the psychological integrity of the patient with Alzheimer's who is living in the community.

This brief and selective review of current projects in Alzheimer's Disease underscores the recent dramatic advances in research knowledge regarding this dreaded illness, while highlighting the broad commitment to the full range of affective, social, psychological, biochemical, and anatomical concomitants of this serious form of brain pathology in the elderly. In a systematically devised, step-wise approach to the development of research understanding, the Institute has stimulated and supported projects focused on: (1) determining the distribution of Alzheimer's Disease in the general population; (2) determining the cause of the disease, and the nature of its clinical course; (3) formulating more reliable methods of identifying and diagnosing Alzheimer's Disease; (4) developing effective strategies for treatment and management; and (5) evaluating the impact of Alzheimer's Disease on its victims, their care givers and family, and the health care system at large.

In view of the extent of mental disorders in old age and the urgent need to increase our knowledge base regarding the most common mental illness of later life, NIMH has developed and implemented a new program of support for Clinical Research Centers on Psychopathology of the Elderly, as a key mechanism for advancing knowledge in the mental and emotional disorders of senescence. The CRC/PE program is intended to provide stable sustained support (of up to $350,000/year for a maximum of 5 years) to a limited number of centers, with an emphasis on bringing basic science advances into the context of patient-oriented studies and clinical application. Because of the devastating impact of Alzheimer's Disease on the elderly and the urgent need for research, the first two centers to be funded are focused on Alzheimer's disease. The CRC headed by Jerome Yesavage at Stanford University, for example, will attempt to identify areas of "excess disability" in Alzheimer disease patients which may be alleviated to improve functional status. The center includes a biochemical component which will attempt to identify biochemical markers which may be useful in differential diagnosis, an electrophysiologic component in order to monitor the natural course of Alzheimer's disease; a sleep component which will screen for sleep-related respiratory disturbance and periodic leg movements in these patients; a medical component which will gather data on endocrine markers and incontinence, and a psychosocial component which will study the presence and course of depressive disorders in family members and caregivers. A second CRC focused on Alzheimer's disease is headed by Burton Reifler at the University of Washington in Seattle. In addition to recruiting subjects, recording their clinical characteristics and following them to autopsy, the investigators will be studying somatostatin related peptides in patients with Alzheimer's disease, and also focusing on the status of several noncholinergic neurochemical systems in Alzheimer's disease which have been implicated as modulators of memory and cognition.

In addition to these areas of research within NIMH, this Institute works closely with several Institutes within the NIH on various collaborative efforts. Our intramural scientists share their ideas and results.

NNIMH and NIH issue joint program announcements, collaborate on the assignment of research grant announcements, and jointly fund research grants.

I. What is the Distribution of Alzheimer's Disease in the Population?

In an effort to examine the distribution of late-life dementias, stratified random samples of the population at major sites across the country are being studied. These studies are designed to assess the level of cognitive functions of elderly individuals living both in the community and in institutions. Rigorous structured interviews, diagnostic instruments, and diagnostic research criteria are being used. Disproportionately large samples of elderly persons have been examined at the Duke University and Yale University sites. Even more intensive psychiatric and neurologic studies are being carried out on the oversample of elderly studied at the Johns Hopkins site in Baltimmore. At the Baltimore site, in addition to complete physical examinations, intensive examinations of brain structure, neurochemistry, and neuropsychology have been carried out on all individuals identified as positive for dementia by Drs. Marshall Folstein and Allan Ramanofsky. These major, carefully planned, systematic epidemiologic studies of randomly selected elderly should provide data of inestimable value to investigators around the world. Epidemiologic studies of this nature are urgently needed to define the prevalence and risk factors associated with this disorder, since available estimates are based on a patchwork of non-comparable studies. Furthermore, most of the currently available information regarding structural, neurochemical, and behavioral differences in the brain functions of Alzheimer's disease patients are based on non-randomly selected samples whose generalizability may be suspect.

II. What Causes Alzheimer's?

New hypotheses in research of the elusive cause of Alzheimer's disease are being vigorously tested in a growing number of studies. These studies focus on etiological factors as diverse as chemical deficiences, brain lesions, autoimmune disorders, and genetic abnormalities.

Over the last decade, as a result of intramural and extramural research, our understanding of the complex processes involved in brain function has advanced tremendously. A major area of progress concerns an enhanced understanding of the mechanisms that neurons use to communicate with each other. Neurons have been shown to release chemical messengers, known as neurotransmitters; the specific effects of these messengers are interpreted by receptors on the adjacent neurons. Because the process of chemical neurotransmission can be manipulated by drugs, it represents an important site for the treatment of symptoms resulting from disorders of the brain. A number of drugs are successfully used in psychiatry to treat disorders that involve abnormalities of chemical neurotransmission; these include neuroleptics in schizophrenia and tricyclics in severe depression, to mention a few.

Over the last several years, a number of investigators with support from the Public Health Serice (PHS) have examined the brains of patients who have died with senile dementia for abnormalities in chemical neurotransmission. Out of that work, two lines of investigation have provided compelling evidence of impaired function of neurons that utilize the neurotransmitter acetycholine in the cerebral cortex. First, a host of studies have revealed that drugs which interfere with the action of acetylcholine in the brain can produce in young individuals memory impairments resembling those in Alzheimer's Disease.

Second, measurements of the enzyme that forms acetylcholine have revealed profound reductions of its activity in the cerebral cortex and related structures in individuals who have died with senile dementia. Investigators have also demonstrated that the neurotransmitters contained in a number of other neurons located within the cerebral cortex are significantly unaffected in Alzheimer's Disease. Accordingly, the chemical deficiency in acetylcholine-utilizing neurons in the cerebral cortex appears to be relatively selective in Alzheimer's Disease.

Until recently, the source of cholinergic innervation to the cerebral cortex remained unclear. In a series of dramatic studies at Johns Hopkins University carried out by PHS grantees Joseph Coyle, Dr. Mahlon Delong, and Dr. Donald Price, it was demonstrated that the acetylcholine neuronal fibers regulating the cerebral cortex come from a group of neuronal cell bodies located in the base of the forebrain in an area known as the nucleus basalis. Selective damage of this area of the brain reproduces in experimental animals the chemical abnormalities in the cerebral cortex observed in senile dementia. Examination of the brains of individuals who died with Alzheimer's Disease revealed that this group of neurons died off selectively.

While the acetylcholine-releasing neurons in the nucleus basalis are not the only neurotransmitter systems affected in senile dementia, nevertheless, they are the most consistently and severly damaged identified neuronal population. These findings are highly significant, and confirm the hypotheses of other investigators,* who are actively testing the hypothesis that enhancement of acetylcholine neurotransmission in the brain might reduce the symptoms of Alzheimer's Dementia.

Ongoing studies are also evaluating other hypotheses regarding the cause and progression of Alzheimer's Disease. The work of Lissy Jarvik, for example, at UCLA Medical Center, is evaluating whether patients with Alzheimer's Disease have a higher frequency of genetic abnormalities and of immune system changes than do older individuals diagnosed with depression or with multi-infarct dementia. Her study will also determine whether such changes can be detected in the first degree relatives of Alzheimer's patients.

In the Intramural program, a series of studies of the genetic transmission of "Alzheimer's Disease" is underway, using a computerized mapping technology which automatically detects protein or gene abnormalities (polymorphisms) in the blood or skin cells. The investigators hope to find errant proteins which might be used as markers in screening for persons genetically predisposed to develop the disease. Additional studies are examining the contribution of aluminum in the developmment or course of the disease.

III. How Can Alzheimer's Disease be Identified?

Clinical research remains complicated by the fact that the state-of-the-art continues to demand that the diagnosis of Alzheimer's Disease in the living patient be formulated solely on the basis of exclusionary criteria. That is, the diagnosis, at present, can only be reliably formulated if all other possible causes of cognitive impairment have systematically been ruled out. Accordingly, a variety of important studies are presently underway, which are attempting to discover new biological or behaivoral markers capable of reliably indicating the presence of Alzheimer's disease in older persons.

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*A selective sample of these projects are described under Section IV, on Treatment Studies

Some highlights of this work include, for example, studies of the elctrophysiologic changes which accompanny the progressive dementing process. The work of Patricia Prinz, at University of Washington (Seattle), is illuminating the nature of the sleep pattern and circadian rhythms in Alzheimer's Disease patients, in an effort to derive more precise indicators of the presence and course of disease. Correlations of anatomical damage of the brain with behavioral abnormalities are being explored through use of computerized axial tomography (CAT scanning) by Ajax George, of New York University in New York City. Studies by Bert Kopell, at Stanford University, and Leonard Berg, at Washington University (St. Louis), have focused on studies of event-related brain potentials, in the hope of using increases in the latency of P-300 as diagnostic markers for Alzheimer's Disease. In a study of EEG sleep parameters, Charles Reynolds of the University of Pittsburgh is working to develop objective differential indicators of the presence of Alzheimer's Disease by relating differences in REM sleep timing and density to defects in acetylcholine production. Exploration of the contribution of positron emission tomography (PET scanning) to the dignosis of Alzheimer's disease is being carried out by Steven Ferris of New York University in New York City.

In an important and exciting new approach which could potentially identify molecular defects associated with Alzheimer's Disease, Carol Miller of the University of Southern California is examining postmortem brain tissue of suspected Alzheimer's patients for specific features using a panel of monoclonal antibodies which were developed to Drosophila nervous tissue, but which recognize human CNS tissue by neurotransmitter enzyme and receptor assays.

In differential diagnosis, studies of memory and cognition are also important, not only because we know little about the clinical course of Alzheimer's Disease, but because mood disorders appear to be early correlates of dementing illness (although they may also closely mimic organic impairment). The more that is known, with precision, about the specific effects of the illness on parameters of memory and mood, the more effective neuropsychological and behavioral tests can be. At the Texas Research Institute of the Mental Health Sciences in Houston, George Niederehe is studying different forms of impairment in Alzheimer's patients and in elderly people diagnosed with depression, with an aim to discovering which memory mechanisms are differentially impaired. Using a different approach, Bart Vrtunski, at Case Western Reserve University in Cleveland, is examining the nature of decision-making and information processing in individuals with Alzheimer's disease to determine whether the locus of the cognitive impairment can be differentially traced to problems in sensory input systems, stimulus encoding, response selection, or response execution stages of choice reaction-time behavior. He hopes to emerge with a battery of empirically-based memory tests which will sharpen the precision of Alzheimer's diagnoses.

In another study intended to further understanding of the nature of memory disorder associated with Alzheimer's Disease, and to establish an efficient, reliable procedure to be employed in early differential diagnosis, Robert Hart of Virginia Commonwealth University is attempting to determine the rate at which recently acquired information is forgotten in patients with Alzheimer's disease, as compared with depressed elderly. Differences in decision strategy and learning efficiency among Alzheimer disease and affective disorder patients will also be explored and documented.

Neuropsychological studies in the intramural program have distinguished the nature of memory loss in Alzheimer's Disease from that seen in Korsakoff's Disease and depression; patients with Alzheimer's have been found to have little access to semantic memory--previously acquired knowledge--and therefore have significant difficulty organizing and encoding ongoing events. In contrast, Korsakoff's patients can access previously acquired knowledge, but show a gross impairment of recent (episodic memory). While depressed patients also show episodic forms of memory failure, these are typically associated with tasks requiring sustained attention.

Findings such as these can go far toward enhancing our understanding of the nature of the cognitive dysfunction in Alzheimer's Disease, and toward assisting in the formulation of new and better approaches for diagnosis and accurate assessment.

Because the diagnosis of Alzheimer's disease in the living patient must be formulated solely on the basis of exclusionary criteria, it is critical that the diagnostic yield and cost effectiveness of a broad spectrumm of diagnostic procedures be systmatically evaluated. Given the growing concern with the rapidly escalating costs of medical care, questions have been raised as to whether the costs of providing older persons suspected of having dementia with exhaustive, comprehensive medical evaluations, running into the $1,000 range, are really worth the outcome. It has also been suggested that such costs are more than compensated for by reductions in the cost of inappropriate treatment or inappropriate placement in nursing homes or long-term facilities. Eric Larson, at the University of Washington, Seattle, is currently evaluating the cost effectiveness of various components of a thorough evaluation designed to detect reversible causes of dementia in the elderly. The finding will result in the formulation of a set of predictors which will yield the most effective differential and prognostic information.

The differential diagnosis between severe depression and Alzheimer's disease is commonly acknowledged to be one of the most difficult and problematic in all of geriatrics. This issue constitutes a significant problem, not only because depression appears to be a common early correlate of dementia in senescence, but also because it may closely simulate a dementing syndrome. The importance of formulating an accurate diagnosis in the elderly is critical, especially in cases of depression mistaken for dementia, where the failure to treat may result in suicide. Although the evidence is clear in suggesting that older persons with depression complain more about their memory than do aged persons with normal mood, the literature is controversial on whether or not individuals with depression actually manifest a measurable memory deficit.

The nature of the association between memory impairment and possible cause and effect in this equation remains questionable. For example, does perceived memory change cause a change in mood state, or is the depression itself responsible for the decline in memory? Is the problem simply amplified by memory complaints? Most important, are there qualitative differences in memory processes between depressed and demented patients that might assist in the distinctions? Evidence of differential effects on these separate memory processes as a function of diagnostic classification may lead to insights regarding the basis of memory problems. The relationship between memory measures and clinical outcome may well yield immediate practical applications by providing possible prognostic indicators.

IV. What are the Most Important New Leads Regarding Treatment and Management of Alzheimer's Disease?

Compelling evidence has accumulated from a number of lines of research suggesting that Alzheimer's Disease is associated with the impaired function of neurons which utilize the chemical messenger acetylcholine in the cerebral cortex. Intramural scientists have demonstrated that drugs which interfere with the action of acetylcholine in the brain can produce in the young, cognitive impairments that resemble those seen in Alzheimer's disease. Scopolamine, for example, which antagonizes the neurotransmitter acetylcholine, has been found to specifically impair the encoding consolidation and retrieval of memories.

On the other hand, drugs that enhance acetylcholine activity (e.g. lecithin and anticholinesterase) appear to produce moderate improvement in memory in some Alzheimer's patients. Currently a number of studies exploring these issues are being supported by PHS. These include, for example, a study of lecithin and piracetam (by Steve Ferris, at Ne York University), in which provocative results from animal studies by Bartus have led to an open clinical trial in eight humans of whom three to four showed some sign of improvement in cognitive function. A new project represents an effort to determine whether this finding can be maintained in a double-blind control trial.

Suzanne Corkin at the Massachusetts Institute of Technology is investigating a variety of cholinergic interventions including lecithin and psysostigmine, a cholinesterase inhibitor. The study is based on work by intramural scientists and others, showing that physostigmine exerts a facilitating effect on learning and memory. Other studies carried out by Tinklenburg, at Stanford, include investigations of the efficacy of neuropeptides in the treatment of Alzheimer's Disease, including ACTH 1 - analogues of the neurotransmitter vasopressin (DDAVP). In evaluating these agents, intramural scientists have found DDAVP to be associated with a small improvement in learning and recall. These improvements, however, were found to be much greater for depressed and normal patients, suggesting that progressive and irreversible destruction of brain cells in Alzheimer's disease severely limits DDAVP's efficacy for dementia patients.

Of interest over the past year has been work of Barry Reisberg and Steven Ferris, at New York University, in focusing on the effects of the opioid antagonist, naloxone, in the treatment of patients with Alzheimer's Disease. Neuroscience advances over the past decade have revealed the existence of natural opium and morphine-like substances in the human brain, and to some extent, throughout the body. These substances known as the endorphins and enkephalins are believed to have important effects on brain functioning. Naloxone blocks the effects of these natural opium-like substances. Preliminary findings by these investigators suggest some mild positive effects in some patients treated with naloxone, which appear to last for 1 to 3 weeks following injection. More extensive studies of the agent are presently underway, however, and these results represent only initial findings. Further research may bring us closer to having a clinically effective treatment for this major illness.

Because of the frequency with which mood disorders appear concomitantly with Alzheimer's Disease, a number of investigators are also focusing on the treatment of depression in the face of chronic, progressive, dementing brain disease. For exammple, Burton Reifler, at the University of Washington in Seattle, is studying elderly patients with dementia, both with and without depression, to determine if they differ with respect to antecedents and response to treatment with a trial of tricyclic antidepressants. Do the two groups have different outcomes regarding cognitive function? Arnold Merriam, at the Albert Einstein College of Medicine is evaluating the presence, nature, and treatment of mood disorder in Alzheimer's patients, in an effort to better understand the course and outcome of pseudodementing and true dementing states, especially when the latter appear concomitantly with mood disorders.

Studies evaluating the utility of behavioral interventions are just beginning, and that area, too, appears to be rapidly developing. In an initial study Steven Zarit, at the University of Southern California, evaluated a variety of operationalized behavioral treatments for elderly persons diagnosed with Alzheimer's Disease who were living in the community, in an effort to specify the differential efficacy of specific behavioral organizing strategies, mnemonic tchniques, and interpretive approaches. He found that in the form in which these approcahes are currently conceptualized, they appear to have minimal impact on memory, but on the positive side, they tend to lessen feelings of depression in the mildly impaired. Working along similar lines, Jerome Yesavage of Stanford University is evaluating the efficiency of enhanced cognitive retraining techniques based on mnenomics for improving declining memory in dementia patients. He is focusing on the impact of these approaches on memory skill, ability to concentrate and subjective complaint of cognitive impairment. The recent advances in our understanding of the basis for senile dementia results from a close interaction between neuroscientists and clinicians. Information derived from basic neurobiologic research is now rapidly being utilized to analyze disorders of the human brain. While it would be misleading to suggest that a cure for senile dementia is upon the immediate horizon, nevertheless, it is clear that over the last several years we have moved from ignorance and therapeutic nihilism to a position of asking informed questions concerning the cause and treatment of this devastating disorder.

V. What is the Impact of Alzheimer's Disease on the Family?

Along with growing concern regarding the serious economic cost of this disorder on the fabric of American life,is the rapidly emerging awareness regarding the devastating emotional impact that dementing illnesses exert on family members and relations. Research by Elaine Brody at the Philadelphia Geriatric Center and by Walter Poulshock of the Benjamin Rose Institute in Cleveland has shown that family members caring for Alzheimer's patients appear to be subjected to severe stresses and may be at especially high risk for depressive reactions. The inability to alter the course of the disease, the nature of the demanding care required, and the inevitable bereavement to be faced make such reactions especially probable. In a study of stress vulnerability in Alzheimer's patients' families, Joseph Becker, at the University of Washington in Seattle, is presently studying the predictive power of two specific models of depressive vulnerability in these families. In a related project, Adrian Ostfeld, at Yale University, is examining the consequences of spousal illnes and death on a surviving partner.

Further research in this area is underway at the Texas Research Institute of Mental Sciences in a clinical and longitudinal study of relatonships between fammily systems and dementia. The study is examining patterns of stress in community caregivers who provide home care to elderly relatives with progressive chronic brain disease. Since dementia is seen as a process which affects all members of the family unit, the study will evaluate how family systemm characteristics mediate the impact of the disease on caregivers and patient alike. Measures of social role functioning, physical and mental health, and subjectively experienced strain over time will be obtained from the primary caregiver and other family members. A variety of current and historical factors in the family system are being assessed as potential predictors of the disease's impact on other family members, and reciprocally, of the patient's own maintenance of function over time. Interpersonal factors, such as traditional family loyalties and past relationship between patient and caregiver, characteristics of the extrafamilial social network, and elements of family cohesion and adaptability, are also being investigated. The study will help to clarify how the family system influences changes in both caregivers and dementia patients over the progressive course of chronic brain disease. Coping patterns and other critical factors which predict better outcomes will be identified.

Other studies include the work of Steven Zarit, at the University of Southern California, who is evaluating the differential efficacy of family and group counseling for caregivers of patients with Alzheimcr's Disease, and developing a means of reducing the burden of care, and making home placement feasible for those families who desire it. In the context of the Epidemiologic Catchment Area Project, Terri Sluss, formerly of Johns Hopkins University, in collaboration with Drs. Ernest Gruenberg and Morton Kramer, is assessing the extent of emotional and economic burden on the caregivers of Alzheimer's patients in a stratified sample of the population in Baltimore. Joanne Steuer, at the UCLA Medical Center, is sytematically studying the impact of support groups for caregivers of elderly patients with Alzheimer's Disease, in an effort to determine their effect on levels of depression, anxiety, and feelings of burden in family members. At Johns Hopkins University, Peter Rabins is carrying out research focused on the delivery of care to Alzheimer's patients and their caregivers.

Ideally the results of this family-oriented research will have important applications for a variety of service providers and particular usefulness to such key connsumer groups as the Alzheimer's Disease and Related Disorders Association. Finally, studies are also underway to determine the special ethical problems entailed in studying Alzheimer's disease patients. The work of Barbara Stanley of Wayne State University in Detroit, for example, is focusing on informed consent issues in such individuals.

In summary, even though the problems in understanding the causes and effects of Alzheimer's Disease, in estimating its distribution in the population and in devising effective treatment strategies for its amelioration remain great, it should be noted that significant progress has been made in moving us from a position of darkness and ignorance to one of asking focused and informed questions in the historic effort and challenge to understand, treat, and prevent this devastating and costly disorder.

Mr. GORE. Thank you, Doctor. We appreciate your statement.

Let me get, for the record, first of all, what your funding is. Dr. Williams, besides the $3.5 million appropriated for fiscal year 1984 to establish up to five Alzheimer's disease research centers, how much other funding does your Institute provide for the study of Alzheimer's disease?

Dr. WILLIAMS. Approximately 15 million other dollars this year for research and training related to Alzheimer's disease and neurosciences in general, but most of that is Alzheimer's disease.

Mr. GORE. What percent of your total budget is the funding for Alzheimer's?

Dr. WILLIAMS. Twelve percent of our total budget.

Mr. GORE. Dr. Goldstein, what is your current funding in this area?

Dr. GOLDSTEIN. Approximately $20 million, and that is approximately 5 percent of our total budget.

Mr. GORE. Same question.

Dr. SILVERS. I have difficulty being that specific, but it is a lot of our basic neuroscience research relates to this disorder. We fund about $9 million in research on aging related to Alzheimer's, and we have specifically $5.7 million targeted to this disorder, but all of the money spent in the basic research and the clinical research and the specific aging research relate to this.

If I could round off and perhaps correct later for the record, I would say about 5 percent for the disorder itself.

Dr. WILLIAMS. I would like to provide accurate figures, too, if you will permit me, because the percentage is too low considering the Center's funding, as well as our total other funding.

Mr. GORE. The total would be in the range of $40 million.

Dr. WILLIAMS. Something like that.

Dr. GOLDSTEIN. That is a ballpark figure.

Mr. GORE. All right. You talked about PET scanners, and Dr. Williams, you referred to them in your prepared statement. If this tool was to be used by other centers in hospitals for diagnosis, is the cost reimbursed by third-party payments?

Dr. WILLIAMS. As far as I know, no. It is entirely a very promising research implement, but it has not been made available, to my knowledge, for simply a diagnostic service.

Do you have any thoughts to add?

Dr. GOLDSTEIN. There are 10 PET scanners in the country. In order to run a PET scanner, one must have a dedicated cyclotron in the hospital, or in the research unit. It costs approximately $3 million just in equipment before one pushes the first button.

It takes a team of up to 30 people to operate the unit. The point I am really trying to make is at this time, PET scanning is only a research instrument, a human research instrument, but only a research instrument, not a clinical care instrument.

There are a few very, very special circumstances in which it is used for differential diagnosis, but those are really quite rare and very special.

This time, again, please let me reemphasize, it is a research instrument.

Mr. GORE. All right, we have read reports that even at autopsy, which you have said is currently the only method by which a definite diagnoses can be made, several patients diagnosed during their life as having Alzheimer's disease turn out not—have had it, and vice versa.

Therefore, it is pleasing to read in your statement that specific criteria for clinical diagnosis of the disease have been established and these criteria have been published, so that they can be uniformly used by physicians to make a more definitive clinical diagnosis.

Later, we are going to be hearing from some of the investigators whose work you are supporting, and I just wanted to make that comment, that is a very definite step forward.

Dr. Silvers, in listening to this testimony, there are a number of similarities described in the type of research being supported, but I take it that precautions are taken by the Institutes to assure that no double funding is given for the same research project, is that correct?

Dr. SILVERS. That is correct. But we do share a neuroscience base, and sometimes the basic neuroscience research can pay off for all the institutes. For example, the deoxyglucose tagging and the mathematical calculations involved in that that have been talked about that allow the CAT scan to be developed, was developed by an intramural scientist at the NIH, Dr. Sokoloff.

But, clearly, all of the extramural funding—there is no double effort. We may collaborate so one institute will pay for one part of the effort and another institute for another part.

Mr. GORE. Very good. You mentioned in your testimony studies in your intramural program to attempt to distinguish the memory loss in Alzheimer's from the memory loss in Korsakoff's disease. Could you tell us briefly what is Korsakoff's disease and how can the two be distinguished by difference in memory patterns?

Dr. SILVERS. I think all three institutes are trying to come up with a better biological tool. The best would be a marker, a certain enzyme or chemical change you could document or physiologic change you could pick up with one of the research instruments initially.

In the meantime, we are looking at some of the behavioral or clinical efforts. We know there are a number of medical disorders that create memory loss. One is a brain disorder created by alcoholism which creates certain changes in the brain and a certain loss of memory and a certain technique which can cope with that, Korsakoff's.

We are trying to do some research in terms of evaluating these memory losses to see if that could be a diagnostic clue, are they different with different populations, not just the recent measurements of recent memory, past memory, but more subtle techniques we can develop.

Mr. **Gore**. We noted in a recent article in the Washington Post, September 14, that one of your intramural researchers is planning a clinical trial with a new drug, deprenyl, for Alzheimer's patients.

Do you hold out some hope for that? What specific benefit is that expected to give to Alzheimer's patients?

Dr. **Silvers**. Most of the pharmacologic research is related to our ideas about the cholinergic system and something that may improve the functioning, naloxone being one reported approximately a year ago.

They are all attempts to try and modify the biochemistry of the area of the brain that we think is involved with Alzheimer's disease, and specifically to try to get this neurotansmitter system elevated in the hope that it would improve memory.

Perhaps Dr. Williams or Dr. Goldstein want to add to that.

Dr. **Williams**. I would just add our research program in the clinical center in Bethesda is also undertaking trials of new drugs. I think they are all in this same general family of drugs, of drugs that will increase the effectiveness of acetylcholine.

Dr. **Goldstein**. I think a model we are discussing is the model that we all have used in terms of another chronic debilitating disease of the brain called Parkinson's disease, which until about 10 or 12 years ago had many of the same horrors that we are discussing in a clinical manifestations of Alzheimer's disease.

The breakthrough was that a neurochemical was found called L-Dopa which helped restore the function of part of the brain. Using that model, our hope is that we can also develop a drug which will restore part of the clinical acuity of the patient.

I think we have to be extremely careful that we don't confuse that with stopping the disease. Of course, in the case of L-Dopa we did not stop the disease Parkinson. What we were able to do is restore functioning, even though the disease is progressing.

And so the materials that I think we are all reading about, hearing about and supporting are, again, in that model, an attempt to see if we can restore function, hopefully perhaps arrest disease.

But we are not that far along yet.

Mr. **Gore**. Now, Dr. Silvers, the New York Times reported that Dr. William Freed at NIMH is exploring some aspects of brain cell transplants with respect to diseases such as Parkinsons and Alzheimer's. Do we have any reason to feel optimism about that prospect?

Dr. **Silvers**. The model is similar to the one we talked about in medication. If an area of the brain is not producing the proper neurochemicals, is it possible to transplant something into the brain that will pick up that function.

Some of the earlier work was with Parkinsonism and did find some improvement.

I think we are hoping that any of these techniques might give us the breakthrough. At the moment, it is giving us basic knowledge and basic research information about brain function.

But I don't know whether I could say yet we have optimism that that is going to turn out to be the cure. Again, this type of research is going on in the other intramural centers as well.

Dr. **Williams**. I might just comment some very exciting work is going on in animals in transplantation of brain cells, where they really—it can be demonstrated they will restore lost function when transplanted into very specific, localized areas of the damaged or aged brain of small animals.

It is far from being ready for human trial. But it holds out some really long-term promise, it seems to me.

Mr. **Gore**. Tomorrow we are going to hear from Dr. Charles Marotta and Dr. Peter Davies. Do you all agree with me that these two findings are extremely significant, or how would you—I mean we want to caution against any, you know, overreaction to new breakthroughs.

The families suffering with the consequences of Alzheimer's disease have enough to bear without false hopes being extended. But how would you characterize these two recent breakthroughs?

Dr. **Goldstein**. I will stick my neck out and say I consider them exciting. They still remain to be demonstrated that they are significant.

Mr. **Gore**. OK.

Dr. **Goldstein**. There is a fine point there.

Mr. **Gore**. I don't have my dictionary with me.

Dr. **Williams**. I would say that the observations are important. They appear to be sound, new observations, that are data we have got to deal with, to come to—be able to experiment and apply. And exactly how to interpret them in terms of the next steps, I think, requires a lot more work.

That is about where I would be on it.

Dr. **Silvers**. As a psychiatrist, I will be symbolic. I think each of these major findings opens a new road to take. We just don't know where the end of the road will lead us.

It may lead us to basic knowledge that will help us in a different disorder, or it will lead to a treatment technique. We just don't know yet.

Mr. **Gore**. OK. Very good. We could obviously ask a lot of other questions.

Would you be willing to respond to additional questions in writing?

In light of the time, we are going to go on to our final panel, but I would like to thank you very, very much for your statements here today.

Mr. **Gore**. Now, I would like to call our third and final panel. Dr. Herbert R. Karp, M.D., professor of neurology, School of Medicine, Emory University; Dr. Donald L. Price, M.D., professor of pathology, neurology, and neuroscience, director, Neuropathology Laboratory, Johns Hopkins; Dr. Charles E. Wells, M.D., clinical professor of psychiatry and neurology, geriatric psychiatry unit, Vanderbilt University; and Dr. Kenneth L. Davis, M.D., Chief, Psychiatry Service, Veterans' Administration Medical Center.

Without objection, the entire prepared statements of this panel will be included in the record in full. We will now proceed with the presentation.

If you care to summarize any portion of your statements, please feel free to do so.

Dr. Karp, we will begin with you.

I want to thank you for being here. We hope that this 3-day hearing will help us and the Congress to determine how we can better focus our research effort and better designed efforts to assist caregivers, and this panel is an extremely important one in pursuit of that goal.

We are glad to have you here. Please proceed.

STATEMENTS OF HERBERT R. KARP, M.D., DIRECTOR, DIVISION OF GERIATRIC MEDICINE AT EMORY UNIVERSITY, AND DIRECTOR OF MEDICAL SERVICES AT WESLEY WOOD CENTER; DONALD L. PRICE, M.D., PROFESSOR OF PATHOLOGY, NEUROLOGY, AND NEUROSCIENCE, DIRECTOR, NEUROPATHOLOGY LABORATORY, THE JOHNS HOPKINS HOSPITAL; CHARLES E. WELLS, M.D., CLINICAL PROFESSOR OF PSYCHIATRY AND NEUROLOGY, GERIATRIC PSYCHIATRY UNIT, VANDERBILT UNIVERSITY; AND KENNETH L. DAVIS, M.D., CHIEF, PSYCHIATRY SERVICE, VETERANS' ADMINISTRATION MEDICAL CENTER

Dr. Karp. Congressman Gore, I would like to amend your introduction by stating that I am at present a director of the division of geriatric medicine at Emory University, and also director of medical services at Wesley Wood Center.

I make this amendment not out of personal vanity, but to respond to something you said earlier in your introduction, to indicate that medical schools are responding, albeit belatedly, to the demographic imperative of an aging population, and to the increasing presence of age dependent diseases, such as Alzheimer's disease.

Mr. Gore. Let me reiterate, I think it is a very understandable and human problem that physicians share with everybody else in the society, that when a new realization takes place, there is just a time lag in adjusting to it.

Now, perhaps physicians and medical educators have a special obligation to minimize that time lag. But——

Dr. Karp. It is beginning to happen. That is what I wanted to bring to your attention. As such, I am really physically located where the problems are occurring, and where questions are being asked.

And in this capacity, I have the privilege of really participating in what has been a truly remarkable convergence of science with a deep concern for the problems of families and the problems of patients and the families of the patients of people affected by this disorder.

In the interests of time, rather than repeating what I have in my prepared remarks, I would like to highlight what we know about the clinical course of Alzheimer's disease, what we know about the epidemiology of Alzheimer's disease, what we need to know, and why we need to know it and how I think we can best fill in these gaps in our knowledge.

First of all, with regard to the clinical course, the mode of the onset of the disease and the details of the early course of the disease are often difficult to establish. The demented patient themselves are really not reliable witnesses and cannot give us these details.

And often the onset and the early course is so subtle as to escape the notice of even the most observant family member or associate. And most often the initial symptoms are noted only in retrospect, after the dementia, after the loss of memory and intellectual function becomes more manifest.

Second is that in this early phase of the disease, the patient becomes more aware of the loss of mental efficiency, and still has insight. Depression frequently appears, and this depression often, in turn, takes its toll on mental function.

The third point I would like to make is that the memory loss is most obvious in recently acquired material, but if you look very carefully at the memory loss, it involves all aspects of memory and cognitive function and to a degree has remarkable sparing of other areas.

The fourth point I want to make is that in the initial phases of the illness, the physical examination, including a detailed examination of other aspects of brain function, is usually normal. There is no disturbance of consciousness, and the patient can be said to be sound of body but have serious impairment of important aspects of brain function.

The personality disorders are seen throughout the illness, and often constitute one of the major aspects of the illness.

It is important for us to realize another point, and that is that standard laboratory tests, including the examination of spinal fluid and blood, are normal. There may be changes in the electroencephalogram, and in the CAT scan, but these are not diagnostic.

The CAT scan is most useful in excluding other causes of dementia, some of which may be reversible, and thus it is an essential part of the initial assessment.

But we should be reminded of the fact that at the present time there is no objective laboratory test which is diagnostic of Alzheimer's disease.

The next point is as the disease progresses, the memory loss becomes more severe, and we begin to encounter other evidence of disturbed brain function. Often speech, though well articulated, is empty of ideas and the patient can be said to have lost the ability to use language effectively.

Patients will frequently have difficulty in geographic orientation, losing their way in the home or the immediate neighborhood, and become unable to use common instruments of daily living such as the telephone, range top, shopping, keeping track of personal finances and putting together a simple meal and other related activities become progressively impaired to the point where the patient realtively early in the onset, in the course of the disease, is totally dependent on the support of others to provide these essential functions.

I mention these in detail to indicate that these are not just vague concepts. We now know that they relate to actual progression of the disease to involve other specific areas of the brain.

You have heard many statements regarding the prevalence of Alzheimer's disease with regards to the epidemiology of Alzheimer's disease. These data are hard to come by.

There is a need that we get harder data. But I would like to break down a figure that we heard earlier, and that is that 4 to 5 percent of the population over 65 has severe dementia.

If we look at the over 65 age group in detail, we can see that it breaks down into figures that are even more impressive and more indicative of the imperative that is before us.

And that is if you look at the segment of 65 to 70, the prevalence of dementia is relatively low, only 1 percent or less. But by age 85 and over, which is the fastest segment growing of the U.S. population, the prevalance rises to over 15 percent.

Now, if this rate of increase is maintained and as life expectancy extends into the tenth decade, into the nineties, it is anticipated by the year 2030 to 2050 there will be an astronomic 45 percent of the population that are likely to develop dementia at some points in their lives.

It is important, also, to consider that if just the onset of the disease or if its devastating effects can be forestalled for 10 to 15 years, Alzheimer's disease would not have the tragic impact that it has today. Otherwise, as has been stated by many others, the illness will become the Nation's No. 1 health problem.

Now, what additional information do we need from the clinical and the epidemiologic standpoint?

We have a great deal of knowledge regarding the classic course of Alzheimer's disease. There are certain aspects of the illness which still need careful documentation.

These include a detailed correlation of the clinical findings with the disorders of structure and function of the nervous system. At present, as has been mentioned, the absolute diagnosis can only be made by tissue confirmation itself by biopsy or at the time of autopsy.

Biopsy is grossly impractical and difficult to justify and the autopsy, of course, is performed very, very much after the fact. Therefore, it is important to be able to document the earliest manifestations of the disease and to correlate this with objective tests in order to recognize the disease at a time when treatment may be beneficial rather than when it is rampant and there is marked destruction of brain tissue.

An example of these types of studies is that of the Laboratory of Neurosciences at the National Institute on Aging, the work that is going on at the Brookhaven Institute where there is detailed correlation of clinical, psychologic, and behavioral findings with the newer techniques of imaging of the brain so we can observe the brain in life rather than at the time of death.

We also need to know the specific nature of the disorders of cognitive function. By this method we can begin to recognize those aspects which are prepared in order to attempt to capitalize on these functions and assist the patient in retaining relative independence for a longer period of time.

We need to be able to distinguish between the dementia of a progressive degenerative disease such as Alzheimer's and the impairment of intellectual function that is seen in depression in the elderly patient. The dementia of the depression is most often correctable with appropriate management.

We are gaining more information regarding the pathology of Alzheimer's disease and the distinction between these changes and the chemical changes that are going on in relationship to those which is regarded as natural aging.

We still need to pursue these points in order once more by recognizing the earliest manifestations and the site of the earliest disease, we would be more likely to identify possible etiologic factors such as toxins or infectious agents and which might be the cause of the disease.

The next point is that we know that most cases of Alzheimer's disease are spontaneous. That is, there is no family history of the disorder. However, we also know that about 10 to 15 percent of the cases of Alzheimer's disease do occur in a familial setting. And in most families this suggests that there is an autosomal dominant pattern of inheritance.

We also know that a significant and a very large number of patients with Down's syndrome, who live into the third and fourth decades of life, are likely to suffer at that point progressive loss of intellectual function and at autopsy show the classic changes of Alzheimer's disease.

So we need to know more about the genetic and other risk factors. In addition to being important in genetic counseling, this will assist us in identifying patients at risk and how can be investigated in carefully designed longitudinal studies in order to refine our understanding of the clinical course and to assist in the search of the etiology.

Now, your ultimate goal, of course, is to identify the causes or the cause of their disorder and to prevent its occurrence. But as we continue this quest, we must with equal vigor continue through careful assessment our questions to identify the day-to-day problems of management which include, to be sure, the biomedical aspects that we have talked about so far, but also the sociologic and psychologic factors which incidentally can very often be effectively modified to the benefit of the patient.

Thank you.

[The prepared statement of Dr. Karp follows:]

Clinical and Epidemiologic Aspects of Alzheimer's Disease

Dr. Herbert R. Karp Professor of Neurology
Director Division of Geriatrics
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Alzheimer's disease is a disorder of the brain which is characterized by a decline in memory and other aspects of cognitive function. It occurs in middle or late life, is slowly progressive over a period of five to fifteen years and results in severe impairment of those aspects of brain function which we recognize as intellectual ability. This decline in memory and mental function is referred to as dementia.

The disease was originally described in 1907 by Alois Alzheimer who was a psychiatrist, neurologist, and neuropathologist. As a clinician, Alzheimer had the clinical acumen to recognize the symptoms as fitting into a pattern which was distinctive and clearly something other than just another form of madness. As a neuropathologist, he had the knowledge and skills to describe the gross and microscopic changes which he found in the brain of the patient at autopsy. His description of the clinical signs and symptoms as well as the structural changes in the brain still constitute the basic clinical and pathologic features of the disease which bears his name.

Early Clinical Manifestations

The mode of onset and the details of the early course of the disease are often difficult to establish. The demented patient is often, by virtue of the nature of the illness, an unreliable witness and the early symptoms may be so subtle as to escape the notice of even the most attentive family member or associate. Most often, the initial symptoms are impaired memory with difficulty in solving common problems, failure to respond to the environment with customary speed and accuracy, and easy distractibility. The patient often turns to mnemonic schemes and reminders in order to function. Family members and business and professional associates are drawn into the patient's life, serving the patient as surrogate memory. This phase of the illness often first becomes apparent in retrospect after the dementia is more manifest. When interviewed by a physician, the patient frequently turns to the spouse or other secondary observers to supply the details of the history. Indeed, this maneuver may be the initial clue to an underlying dementia. As the patient becomes more aware of the loss of mental efficiency, depression frequently appears, the depression in turn taking its toll on the mental function.

The memory loss is most obvious in recently acquired material, particularly those items which are of an impersonal nature. Careful examination however discloses that this impairment involves all aspects of memory to a varying degree.

In the initial phases of the illness the physical examination including a detailed examination of other brain function is usually normal. There is no disturbance of consciousness. It may be said that the patients are sound of body but have serious impairment of brain function. Disorders of gait, limb paralysis, seizures, urinary incontinence may occur but are characteristically much less prominent and appear later than does the loss intellectual function. Some personality disorders are present to a degree throughout the illness. Although euphoria and hyperactivity may be seen, the most frequent pattern is depression with or without periods of agitation, leading eventually to extreme apathy and withdrawal. The patient deteriorates slowly over eight to ten years to a stage of complete helplessness.

The attending physician must be as certain as possible that there are no systemic diseases or other organic or psychogenic brain disorders which might account for the impairment of memory and cognitive function. The clinical evaluation must include a detailed inventory of all medications which the patient may be taking as well as a through study of possible interactions of medications and their untoward side effects. Any medications which are suspected of affecting memory and cognitive function should be discontinued and the patient observed without medications before a final impression is formed.

Standard laboratory test including the examination of cerebral-spinal fluid and blood are normal. There may be changes in the electroencephalogram but these are not diagnostic. The CAT scan is usually normal for age initially but later may show evidence of loss of brain substance which is referred to as brain atrophy. The CAT scan however is most useful in excluding other causes of dementia some of which may be reversible. Thus it is an essential part of the initial assessment.

As the disease progresses the memory loss becomes more severe and the patient begins to lose other features of brain function. Although there is preservation of the ability to speak often with a remarkable retention of vocabulary, syntax and rhetoric, the patient will frequently have significant difficulty in naming objects. The speech, though well articulated, is often empty of ideas and the patient can be said to have lost the ability to use language effectively. Patients frequently will have difficulty with geographic orientation, often losing their way in the home or immediate neighborhood. They become unable to use common instruments of daily living such as the telephone, rangetop or even the simplest of cooking utensils. Shopping, keeping track of personal finances, putting together a simple meal, and other related activities become progressively impaired to the point where the patient relatively early in the course of the illness becomes totally dependent on the support of others to provide these essential functions.

The family, in addition to attempting to supply these impaired activities of daily living has the added impact of the patient's nighttime sleeplessness, wandering and having to care for such basic functions as toileting, personal hygiene, dressing and feeding.

At variable points in the course of this illness families reach the maximum level of their ability to cope with the illness and turn to other sources of support. They do so often with preconceived conclusion that the patient needs to be admitted to a long-term care facility.

Epidemiologic Aspects of Alzheimer's Disease

Exact data on the prevalence of dementia in general and Alzheimer's disease in particular are not available. However, by combining estimates of the prevalence of dementia in the northern European population age 65 or older with projections from the National Center for Health Statistics, it is estimated that in the

United States 4-5 percent of the population over 65 has severe dementia. An additional 10 percent has mild to moderate impairment of mental function. These numbers are even more impressive when related to specific segments of the population over 65. For example, in the 65 to 70 year age group, the prevalence of dementia is only 1 percent or less. By age 85 and over, the fastest growing segment of the United States population, the prevalence rises to over 15 percent. At this rate of increase, as the life expectancy extends into the late 90's, it is anticipated that by the years 2030-2050 an astonishing 45 percent of the population is likely to develop dementia at some point in their lives!

Based on pathological criteria at autopsy, the largest group of patients with dementia show the changes of Alzheimer's disease. The second most frequent cause of dementia is arteriosclerosis of the cerebral blood vessels. The third is a combination of Alzheimer's disease and arteriosclerosis. The obvious conclusion from these studies is that Alzheimer's disease is the most frequent cause of dementia in adults. These data suggest that there are upwards of one million cases of Alzheimer's disease in the United States today. It has been further estimated that Alzheimer's disease accounts for 40-50 percent of nursing home admissions. Based on the current prevalence and frequency of Alzheimer's disease, and assuming that there will be no effective way to prevent or treat the condition, the prevalence of Alzheimer's disease will triple by the year 2050. It is important to consider that if the onset or the devastating effects of this dementing illness can be forstalled by ten to fifteen years, Alzheimer's disease would not have the tragic impact that it has today. Otherwise, as has been stated by many others, dementing illnesses-particularly Alzheimer's, will become the nation's number one health problem.

The data regarding the financial impact of Alzheimers disease are difficult to obtain. However it is estimated that in the United States today the cost for maintenance in long-term care facilities alone ranges from eight to ten billion dollars per year. For every patient who has "formal care" in an established health facility, there are two or three who have "informal care" delivered by members of the family or other sources of support. A conservative estimate is that it consumes approximately nine hours per week just to perform routine household chores for the demented patient. As the disease progresses to the point that the patient needs help with feeding, bathing and dressing, this burden of support increases to 27 hours per week. Based on these figures it is estimated that in 1978 approximately ten billion dollars of equivalent time was consumed by informal assistance and that in 1982 this figure rose to 18 billion dollars.

The Need for Additional Data Through Clinical Research

Though we have a great deal of knowledge regarding the classic course of Alzheimer's disease, there are certain aspects of the illness which still need careful documentation. These include detailed correlation of the clinical findings with the disorders of structure and function of the nervous system. At present, the absolute diagnosis can only be made by tissue confirmation either by biopsy or at the time of autopsy. Biopsy is grossly impractical and difficult to justify. The autopsy is performed very much after the fact. Therefore it is important to be able to document the earliest manifestation of the disease and to correlate this with objective tests in order to recognize the disease at a time when treatment may be beneficial rather than when it is rampant with marked destruction of brain tissue.

We need to know the specific nature of the disorders of cognitive function in greater detail. We also need to recognize those aspects which are preserved in order to capitalize on these functions and assist the patient in retaining relative independence for a longer period of time.

We need to be able to distingush between the dementia of a progressive degenerative disease such as Alzheimers and the impairment of intellectual function that is seen in depression in the elderly patient. The dementia of the depression is most often correctable with appropriate management.

We are gaining more information regarding the pathology of Alzheimer's disease and the distinction between these pathologic changes and those of what is regarded as normal aging. These studies of structual changes as well as the changes in the neurochemistry of the brain have to be pursued further in order to answer the basic question as to whether this is a disease process per se or whether it represents accellerated aging. The conventional wisdom at present favors the former alternative. By recognizing the earliest manifestations of the disease we are more likely to be able to identify etiologic factors such as toxins or infectious agents which might be the cause of the disease.

We know that about 10 to 15 percent of cases with Alzheimer's disease have a familial incidence. In some of the families this suggest an autosomal dominant pattern of inheritance. But we need to know more about the genetic and other risk factors. In addition to being important in genetic counseling, this will assist in identifying patients at risk who could be investigated in carefully designed longitudinal studies in order to refine our understanding of the clinical course and to assist in the search for an etiology.

The dementing illnesses including Alzheimer's disease make tremendous demands on families and other sources of support. Therefore it is becoming increasingly important to explore other methods of health care as alternatives to long-term care facilities. This would include more liberal use of and financial support for day care, respite care and community oriented care such as block nurses and similar plans of community and self-care.

There is a need for more careful documentation of the full personal and financial impact of Alzheimer's disease with a view toward drafting legislation which will redefine the extent of third-party payment as well as public assistance.

Mr. GORE. Thank you very much.

We will now hear from Dr. Donald Price, director of the neuropathology laboratory, Johns Hopkins.

Dr. PRICE. Thank you very much.

I was asked to discuss a bit about the structural and chemical pathology of the brain. To do that, I would like to resort to some visuals. With the chairman's permission, may I show some slides.

Mr. GORE. Absolutely.

Dr. PRICE. First of all, Alzheimer's disease is not a disease of the 20th century. It has been known for many years. This is a fragment of a poem by Sophocles, the Athenian playwright/poet, who early documented the clinical syndrome of Alzheimer's disease in these two lines.

As an aside, Sophocles himself did not suffer from Alzheimer's disease, I don't believe, in that he wrote Oedipus Coloneous in his 92d year. Before talking about what happens to specific systems within the brain in Alzheimer's disease, I think it might be useful to briefly review certain aspects of brain function.

This is, let us say, my brain. So this is the front, back, down, up. This is the frontal lobe, the parietal lobe, the occipital and the temporal lobe, all terms you will hear over the next couple of days.

Dr. Goldstein this morning mentioned a bit about CAT scan, and that, as I move my right hand, the region controlling the hand area lies here in the left hemisphere, and in the CAT scan, as I do this, or as I talk, this region will light up.

The point is that specific parts of the brain and specifc cells within specific parts of the brain have specific functions. And as you will see, Alzheimer's disease attacks certain specific cell populations in specific brain regions.

Now, we superimpose on that image of the brain a series of neuronal circuits. That is, each color represents a group of nerve cells—the bodies here or here or here give rise to long processes going to other parts of the brain.

As was mentioned earlier, brain cells communicate with each other in the following fashion. Let's say that this is one brain cell and this is another brain cell. And this is, let's say, brain cell A, this is brain cell B. A communicates with B like this.

A is excited, it gives rise to an electrical impulse, and when that electrical impulse reaches my left hand, which is a nerve terminal, a chemical transmitter is liberated, crosses, and it excites or inhibits nerve cell B.

And you will hear today and tomorrow about a variety of neurotransmitter compounds that are either affected or spared in Alzheimer's disease. This diagram is color coded to show some of the circuits which we now know are at risk. This yellow region is the so-called basic forebrain system, which provides the major so-called cholinergic innervation of the cortex.

These cells make acetylcholine, and when it is liberated in the cortex, the cortex is excited. There are other systems, ones that use monoamines or peptides. You will hear about those a little later, and also tomorrow.

The point is that the brain is made up of multiple assemblies of cells that do different jobs. In Alzheimer's disease, certain groups of cells are affected while in Parkinson's, other groups of cells are affected. And the differences in the clinical presentations of the disorders are due to the involvement of different cell populations.

Now, this is a brain of a patient with Alzheimer's disease. It actually doesn't look terribly abnormal. The orientation is very much as I showed you before. This is the front, the top, the back.

So this is occipital, frontal lobe, and temporal lobe. This was actually a middle-aged woman from Boston who had about a 5-year history of a progressive loss of memory, very abnormal behavior, and gradually ended up in a nursing home.

When you look at the brain, there is some atrophy here and here, but it is not really terribly striking. The question is given this brain, what are the reasons for the pronounced failure in the cognitive factors?

When you look at the brain of such a patient, and this could very much be the patient that Alzheimer himself described in 1907, you see three changes, two of which are shown on this slide. And you will hear more about this from other investigators later this morning and tomorrow.

But let me point out two of the principal features of the disease. These triangular structures are the so-called neurofibrillary tangles, which are mentioned in my report—Herb alluded to them, other people will allude to them tomorrow. These structures represent nerve cells that have accumulated unusual filaments that are highly insoluble, and it is as if within the nerve cell there is a tangled skein of abnormal constituents, and intuitively, if a nerve cell is filled with something like a tangled ball of yarn, it probably is not working properly.

The other element is the so-called senile plaque. These are areas in which when nerve cell A communicates with nerve cell B, normally there is a nice connection. In the senile plaque, the nerve terminal of nerve cell A is very, very abnormal, and the communication between nerve cell A and target cell B is very impaired in these specific sites.

And so these two features described by Alzheimer and by other investigators are two of the histologic hallmarks of the disease.

The third, not shown here, is the loss of certain cell populations. In Alzheimer's disease, certain groups of nerve cells disfunction and eventually go on to die. One such system is the basal forebrain cholinergic system.

It should be said this is not the only system involved in this disease. This is the one that from a neurochemical and neuropathological point of view is most consistently involved, but it is not the sole cause of the disease by any stretch of the imagination.

Now, how do we know these kinds of things? First, a neurochemist back in the mid-1970's, and several groups in England, showed that there were in the brains of individuals with Alzheimer's disease a loss of markers for the system that uses acetylcholine, the yellow system I showed you.

And then people began to look at the brains of those patients and look at the cells that gives rise to this cholinertic innervation of the cortex. This was such a case.

This was a 74-year-old, actually, Washington Government worker who at 60 years of age began to develop cognitive deficits, impaired memory, poor judgment in his work. The symptoms progressed and he eventually died at 74 years of age.

So his early onset, 60 years, long clinical course, 14 years, and he has a positive family history of disease. I think increasingly we are recognizing that many of these patients have positive family histories. And that will be amplified tomorrow.

Now, this is the brain of a control patient, looking at that basal forebrain cholinergic system. You can see the number of cells there is quite substantial. However, in the affected individual, the index case that I described, the number of cells there is remarkably decreased.

I use that only as an example. I think there is abundant evidence that there are other systems involved in this disease. So it is not a simple disorder that just by using manipulations of the cholinergic system those patients are not going to be necessarily substantially improved.

There are abnormalities of the monoaminergic system shown here. There is a paper amplifying on the work of Mel Ball from Canada showing nerve cells in the hippocampus are also involved in the disease. So we are not dealing with a simple disorder.

In addition, it might also be said that we now know that Alzheimer's disease occurs in other settings. About 50 percent of people with Parkinson's disease eventually become demented. And many of those, if not all those individuals that become demented, have Alzheimer type pathology, and their mental problems are not made better by L-Dopa.

And then to amplify again the point made by Dr. Karp, why show a picture of a person with Down's syndrome. As Herb mentioned, if this youngster grows up to be 40 or 45 years of age, he will develop the pathologic and chemical stigmata of Alzheimer's disease.

There must be an important clue there. Why with extra chromosomes do you get Alzheimer's disease?

Here I have just mentioned some possible etiologic factors. They will be amplified by investigators tomorrow. And I will be happy to put them in some sort of context in questions. But I don't want to go into them all.

The point is that some have thought aging is important, head trauma, genetic factors, Down's syndrome, abnormalities in the 21 chromosome, aluminum and silicon have been suggested.

Witnesses tomorrow will talk about the role of transmissible agents. And the work of Marotta, and other pieces of work like Marotta, suggesting there may be altered enzymes in the disease, are all I think very important and should be discussed by the investigators who have made those discoveries.

Finally, I would like to say that where I see, we are at present with Alzheimer's disease, we are beginning to understand some of the cellular substrates of the clinical syndrome. But we clearly need much more information. And you will hear over the next day and a half about animal models.

I want to sort of close on making the point that many of these disorders, and many of the degenerative disorders of the nervous system really are rather difficult to study in living patients for a variety of reasons.

And they cannot be studies really in a homogenate or in a tissue culture dish. We really need to examine the brains and then we need to try to make animal models of those disorders. And a number of the people in this room who you will hear from are really expert at making animal models of neurologic diseases.

And I think that that is an area of research that greatly needs emphasis.

Finally, I would like to amplify on Dr. Karp's point. At present if you read the literature, and this is from Parade magazine, you realize that right now there are more people over the age of 65 than there are teenagers.

This is the first time in U.S. history that we have had such a situation. You read the Wall Street Journal and you see depicted the point Dr. Karp made. This shows millions of people over 85 years of age and this shows decades.

So we are sitting here. Our children will be out here. And I think the magnitude of the problem, given that perhaps 15 percent of these individuals will have Alzheimer's disease, is obvious. And it seems to me if we don't make progress in understanding the cellular basis of this disorder, the problems that we pass on to the next generation are going to be formidable.

I will close on that.

[The prepared statement of Dr. Price follows:]

Report for the U.S. House of Representatives
Committee on Science and Technology
Subcommittee on Investigations and Oversight

ALZHEIMER'S DISEASE

The central and peripheral nervous systems are composed of complex assemblies of nerve cells (neurons) which communicate information by electrical and chemical signals. Neurons directly influence other neurons by liberating specific neurotransmitters which excite or inhibit target nerve cells. Specific types of neurons in distinct regions of brain use different neurotransmitters, and each group expresses specific functions. For example, cholinergic neurons in the basal forebrain give rise to projections to the cortex; by liberating acetylcholine, these projections excite cortical target cells. Degenerative neurological diseases, including Alzheimer's disease (AD), Parkinson's disease, and amyotrophic lateral sclerosis, selectively affect specific neuronal populations — a phenomenon referred to as "selective vulnerability". The most common disease of this type is AD. Elderly demented individuals represent more than 10% of the population over 65 years of age, and approximately 50-60% of these patients suffer from AD. As our aged population triples in the next 75 years, the costs for caring for affected individuals will rise at dramatic rates, posing enormous ethical, economic, and social burdens.

Clinical Manifestations

Although dementia occurring in old age was clearly described in ancient times, the disease was not documented in the medical literature until 1907, when Alois Alzheimer described clinical and pathological findings in a middle-aged woman with a progressive dementia. AD frequently starts with gradual loss of memory, followed by progressive deterioration of thought, judgment, and the ability to manage personal affairs. Many patients have problems with language skills, praxis, and visual-spatial perceptions. Eventually, these individuals become severely demented.

Diagnosis

At present, there are no laboratory studies specific for AD. The diagnosis is one of exclusion, and a definitive diagnosis can only be made at autopsy. In the future, new imaging techniques, which allow examination of regional changes in metabolism and transmitter-specific circuits, should provide new insights into the relationships of clinical symptoms and brain abnormalities.

Research on Brain Pathology

Significant progress has been made in identifying changes in neurotransmitter systems in the brains of individuals with AD and in demonstrating relationships between these neurotransmitter abnormalities and histological hallmarks of the disease, i.e., neurofibrillary tangles, senile plaques, and loss of specific populations of nerve cells.

Alterations in Populations of Nerve Cells

Changes occur in a variety of neurotransmitter circuits, including cholinergic, somatostatinergic, and monoaminergic systems. Neurons within the basal forebrain provide the major cholinergic innervation of cortex and, in AD, dysfunction and/or death of these neurons appear to be responsible for reductions in forebrain cholinergic markers. Somatostatin levels in cortex are reported to be decreased in AD, and some intrinsic cortical neurons degenerating in this disease may use this peptide as a neurotransmitter. A number of patients with AD show changes in monoaminergic systems. Degenerative changes also occur in nerve cells of the amygdala, hippocampal formation, and neocortex, but the transmitter specificity of these affected neurons has not been clarified.

Neurofibrillary Tangles

In AD, cell bodies of certain neurons often contain neurofibrillary tangles, consisting of accumulations of unusual cytoskeletal elements. The presence of abnormal filaments may alter the dynamics of intracellular transport processes and have profound consequences on neuronal function.

Senile Plaques

Located in the cortex, these abnormal structures are made up of enlarged axons, synaptic terminals, and amyloid; plaques represent foci of abnormal interactions between neurons. It is not surprising that, when sites of chemical communication between nerve cells are disrupted, affected individuals show dementia. Until recently, little was known about the transmitter specificity of elements in senile plaques. Recent studies indicate that cholinergic, somatostatinergic, and monoaminergic systems as well as, possibly, other neuronal populations participate in the formation of senile plaques.

Experimental Studies in Animal Models

Animal models are extremely useful for investigating important questions raised by studies in AD. Several examples illustrate this point. Macaques with experimental lesions of the basal forebrain cholinergic system (e.g., one of the abnormalities in AD) show impaired performance on certain memory tasks. Aged monkeys, who have memory deficits similar to those occurring in aged humans, show cortical senile plaques. Recent studies have demonstrated that neuronal elements in plaques are derived, in part, from neurons in the basal forebrain cholinergic system, the brainstem monoaminergic system, and several intrinsic cortical systems, including somatostatinergic neurons. Thus, the use of animal models has greatly helped to clarify the consequences of specific neuronal injury on behavior and the roles of specific transmitters in plaques, similar to those occurring in AD.

Etiology

Genetic/Chromosomal Abnormalities

In some families, AD appears to be inherited as an autosomal dominant disorder. First-degree relatives of an affected proband have an increased chance of developing the disease. Relatives of AD patients have increased incidence of Down's syndrome. Moreover, individuals with Down's syndrome who survive into the fourth decade frequently develop the dementia and brain abnormalities of AD.

Infectious Agents

It has been suggested that AD may be caused by a transmissible agent because of certain resemblences between brain abnormalities occurring in AD and those occurring in Creutzfeldt-Jakob disease (a dementia syndrome in humans) and scrapie (an encephalopathy in sheep), both of which are caused by transmissible agents with unique physical/chemical properties. However, these transmissible agents have not yet been demonstrated in the brains of individuals with AD, and a report of experimental transmission of the disease to primates has been retracted.

Toxins

The roles of external toxic agents in AD are not clear. Original reports of increased aluminum concentrations in the brains of patients affected by AD have not been confirmed. Aluminum is present in increased amounts in the nuclei of some neurons showing neurofibrillary tangles, but it is not known whether this change is a primary abnormality or a secondary phenomenon with aluminum deposited in already damaged neurons.

Therapy

At present, there is no specific treatment for the cognitive impairment in AD, although supportive care and management of secondary symptoms can significantly improve the quality of life for both patient and family. A variety of therapeutic approaches has been tried in attempts to correct the cholinergic deficiency. These include: attempts to increase available acetylcholine at synapses by providing precursors, such as choline and lethicin; efforts to slow down the breakdown of acetylcholine by using cholinesterase inhibitors; and direct stimulation of cholinoceptive neurons with muscarinic agonists, such as arecholine. Although some improvements on specific neuropsychological tests have been demonstrated with these approaches, none of these therapies have been reported to consistently improve skills necessary for daily living and, thus, they do not appear to be highly useful in a clinical setting.

The development of new therapeutic strategies for AD will be enhanced by further knowledge of transmitter populations at risk in individual patients and by understanding the consequences of these neuronal changes on the behavior of the patient. For example, new therapies may be designed to enhance (by drugs or trophic factors) or replace (by neural grafts) functions of remaining neural circuits. A better definition of the nature of degenerative processes (i.e., which neurons die when in which patients) will also likely lead to better understanding of the causes and mechanisms injuring these groups of neurons. With this information, we will be in a better position to deal with the approaching epidemic of AD.

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Mr. **Gore**. Thank you very much. That is a very enlightening statement. We appreciate it.

I note for the record that some of the items you mentioned in your statement were first discovered by you.

Let me call now on Dr. Charles Wells from Vanderbilt University.

Again, I hope others will forgive me for expressing a special welcome to Dr. Wells. As a fellow Tennessean, I am proud that when we did our survey of who ought to be included in this panel, there was widespread agreement it would not be completed without your testimony, Dr. Wells. We are delighted to have you here. I invite you to proceed.

Dr. **Wells**. Thank you.

I am grateful for the opportunity of talking with you today about the serious behavioral changes that so often accompany Alzheimer's disease and about the serious problems families and physicians come up against in trying to care for these persons who have Alzheimer's disease.

I am a practicing physician, a psychiatrist and neurologist who specialized in treating elderly patients with psychiatric problems; thus, caring for patients who have behavioral problems due to Alzheimer's disease makes up a major portion of my professional activities.

I believe that I can convey to you the full impact of these problems by telling you about the problems of a few specific patients and their families rather than by simply cataloging for you the full list of possible complications.

Please bear in mind that these examples that I bring you are common rather than unusual. As difficult as they appear, they typify the problems I, my patients, and their families encounter on an everyday basis.

I am thus trying to bring this down to the human dimension, though I must admit I cannot tell the human dimension in terms as meaningful as those used earlier by Mrs. Burns, Mr. Shugart, and Mrs. Grashot in their testimony.

The first patient was an elderly man, who had for several years experienced slow and progressive loss of memory and intellectual functions characteristic of Alzheimer's disease.

Then, for no apparent reason, his course turned precipitously downhill. This confusion and disorientation worsened rapidly, he became incontinent, and he lost the ability to assist at all in his daily care.

He became morose and tearful, began to talk of death, quit eating, and lost weight. For a while his wife, who was devoted to his care, could see no alternative to institutionalization.

A careful diagnostic appraisal focused, however, on the importance of depression in precipitating his rapid decline, and treatment for the depression led to remarkable clinical improvement, even though it did not reverse his intellectual impairment, so that his wife was once again able to care for him at home, which she did until he died of unrelated medical problems about a year later.

Another patient was a woman in her early sixties whose Alzheimer's disease was marked most strikingly by problems in language functions, both in understanding what was said or written to her, and in communicating with others either verbally or in writing.

Since she could not understand either what was happening to her or what she was told, she became anguished and walked about her home and later the hospital for days on end with an expression of almost unbearable distress on her face, so much that we sometimes feared she might also be in physical pain, even though we could find no cause for this.

She became suspicious of others and fearful that they might hurt her, and she would occasionally strike out at others in her bewilderment. Although she appeared more comfortable when she took certain medications, because of her suspiciousness she would take them only sporadically.

Finally, a long-acting medication which could be given by injections afforded her some relief and made her care in a less restrictive environment feasible.

A third patient, a woman of unusual culture and refinement, had lived alone successfully for some years following the death of her husband, and even through the early stages of her Alzheimer's disease.

As her condition worsened, however, she lost the ability to recognize herself in the mirror, and she developed a fixed delusion that the person whom she saw in the mirror was an intruder, a woman of evil intent fixed upon harming her.

Her conviction was such that she went about her house in great fear, lest she encounter that evil woman and be molested by her. She was helped only slightly by medications, and finally her family had to resort to draping or removing all mirrors in the house and keeping a companion with her constantly to reassure her in her distress.

Another patient, an elderly woman with Alzheimer's disease, managed to live alone and take in roomers during the early phases of Alzheimer's disease. Gradually, however, her increasing incapacity and peculiar ways alienated her roomers, and she was left alone.

She was a fiercely independent woman who rebuffed efforts of neighbors and others to help her, yet she was unable to see to her own nutritional and environmental needs.

Her house grew cold and filthy, food rotted on the shelves of her kitchen, and she forgot to feed herself, being beyond the awareness of hunger. All outsiders were suspected of plotting her harm, and those who tried to cross her threshold were threatened with bodily harm.

Finally, the police were prevailed upon to bring her to the hospital, where the results of her physical neglect could be corrected and she could receive adequate care.

The last patient whom I shall describe, an elderly man with Alzheimer's disease, had been an extraordinary athlete in his youth and a successful businessman.

As his Alzheimer's disease progressed, he began to lose his ability to recognize his wife of 50 years or to recognize the home where they had lived together for many years.

He began to see his own home as a strange and unfamiliar place where he was kept a prisoner and where his wife was his guard.

He grew obsessed with thoughts of going back "home," by which he meant the home where he had grown up, and to this mother, who had been dead for many years.

His restlessness and irritability grew with his intellectual decline, and he threatened and even attacked his wife and family when they sought to restrain him. He retained his athletic agility and strength, and on several occasions he managed to evade the locks and other restrictions devised by his family to protect him, only to be found wandering miles away several hours later, once in the midst of an ice storm.

But you have heard enough examples, I trust, to convince you of the importance of psychiatric symptomatology and behavioral changes in Alzheimer's disease.

I could go on for hours with one example after another, and each would be in some important features different from all the others and each in some ways like them.

What points are then to be made? Let me try to make five.

Point No. 1: There are patients whose behavioral changes and psychiatric symptoms are part and parcel of their Alzheimer's disease. None of the patients whom I described to you had behavioral or psychiatric abnormalities prior to the advent of their Alzheimer's disease.

Indeed, patients with Alzheimer's disease have by history experienced no greater psychiatric dysfunction before they developed Alzheimer's disease than has the general population.

Yet, behavioral changes and psychiatric symptoms are important features in almost every patient with Alzheimer's disease who is followed over time, and often they present the most serious problems in patient care and management.

Point No. 2: These behavioral changes and psychiatric symptoms associated with Alzheimer's disease cover virtually the entire spectrum of psychiatric symptomatology—depression, anxiety, worry, fear, paranoid suspiciousness, insomnia, delusions, hallucinations, agitation, restlessness, withdrawal, assaultivenes—although admittedly the spectrum may be narrow in the individual patient.

Point No. 3: These patients suffer, some intensely, as a consequence of these manifestations of Alzheimer's disease, and their families suffer because of the suffering.

Because patients with Alzheimer's disease so often appear unaware of their loss in mental functions, it is easy to think that they must be comfortable, since they are oblivious to what is happening to them.

Indeed, this is probably true in some instances. In the patients whom I described to you, however, and in so many others like them, the suffering in them and in their families is intense, and most of it stems from what might be called the psychiatric manifestatons of Alzheimer's disease.

Point No. 4: There are in this country virtually no systems of the health care delivery and no medical facilities designed for the long-term treatment and care of such patients.

Their problems are such that they often overwhelm even those families most intent on continuing to care for them at home, even when the logistical and financial problems of home care have been overcome.

Most nursing homes, where so many patients with these problems end up, are not designed and staffed properly to provide the care needed. State hospitals, once the last refuge for patients with such problems, have almost totally closed their doors to such patients.

Point No. 5: These patients with Alzheimer's disease, that is, those in whom behavioral changes and psychiatric symptomatology are prominent, can often be helped by high-quality, specialized medical intervention.

These specific manifestations of Alzheimer's disease often respond well to treatment with the therapeutic instruments that are currently available to us.

Even if we do now lack a specific treatment for Alzheimer's disease itself, even if we do still lack tools that will improve memory and other cognitive functions predictably and significantly, we can still aim for and often achieve symptomatic improvement in this group of patients.

My last point, then, is that these patients as a group provide us a classic example of a situation in which we can already treat and help the patient and the patient's family, even if we cannot treat the underlying disease itself.

In other words, we can treat the patient, even if not the disease.

Thank you.

Mr. GORE. Thank you very much.

Our final witness of the day is Dr. Kenneth L. Davis of the Veterans' Administration Medical Center. We are delighted to have you, Doctor, and please proceed.

Dr. DAVIS. Today, I am going to talk to you about the research we have been conducting for development of a treatment for Alzheimer's disease.

As has been stated today, there is no current treatment for the disease, but developments that have occurred in the last 8 or 9 years give us some optimism that at least it may be possible to do better than what we have been doing in the past.

That optimism, perhaps, we might say, we might be a little less optimistic today in 1984 than we were in 1977, when that discovery was made, but that stemmed from an extraordinary development that occurred in three laboratories in the United Kingdom.

That was the elucidation of the neurotransmitter abnormality that we now know to be the most consistent abnormality, cell that make acetylcholine, which is reduced in the brains of patients with Alzheimer's.

We now know today there are other transmitters and abnormalities in the disease, but the cholinergic abnormality is consistently found in all Alzheimer's patients, and undoubtedly any pharmacotherapy will have to deal with that abnormality, though it may have to deal with others as well.

Once that was elucidated, a number of groups simultaneously proposed that we should try to treat this disease by reversing a cholinergic deficit. We were particularly suited at the Mt. Sinai Hospital, of which I am a part, we were suited to proceed with that work, because we had been investigating a drug called physostigmine.

It increases the amount of acetylcholine in the brain, and it does so by inhibiting the enzyme that breaks it down.

The enzyme which breaks it down is acetylcholinesterase, and by blocking the activity of the enzymes, we can increase the amount of acetylcholine in the brain.

We were developing studies with physostigmine in normal young people prior to the advent of these studies in the United Kingdom.

In order to see whether we could make normal young people learn a little more and a little faster, to our surprise, we came up with a science fiction-like result that physostigmine, which given in a controlled double blind vigorous protocol made young, normal people learn a little more a little faster.

That finding, which appeared in Science magazine, was replicated and appeared along with another study from the National Institute of Mental Health, which used a different drug, but also increased cholinergic activity, and found the same thing, which is to say, this they could make young people learn a little better.

The conclusion of both those articles was rather interesting in the parallelism. Both said that these studies with normal people and cholinergic drugs had obvious implications with the treatment of Alzheimer's disease.

It would have seemed, at least at that time, 1978, a lot simpler than it seems now, but what we did was rather than administer physostigmine, or as the people at the NIH had done, arecoline, a number of us began to administer precursors of acetylcholine.

A few years ago, we were all reading about lecithin and choline.

A number of studies were conducted in good places and the results were consistently negative. We now know today that although those drugs or agents are, in fact, precursors of acetylcholine, they don't increase cholinergic transmission, the ability of a cholinergic nerve to talk to another nerve.

We have to turn back to the drugs we knew something about, the drugs we felt were a little more toxic, but at least drugs that we knew could predictably increase cholinergic activity, and we began a study with physostigmine.

We did so with Alzheimer's patients with trepidation, because the drug could have a toxic effect on cardiac function.

There were also other limitations of the drug. It is short acting, given intravenously; and it is difficult to assess change for a drug that may only have 20 minutes of activity.

Nonetheless, the results of our study with intravenous physostigmine were, indeed, we were able to find a statistically significant improvement in the ability of our Alzheimer's patients to learn new information, and since that is one of the core systems of the disease, we were encouraged enough to go on with further studies.

Now, these findings with intravenous physostigmine were hardly as robust as we might have wanted, but nonetheless, in the right direction.

The problem physostigmine in that form, it was very brief acting, and there ensued about 2 years of correspondence between my laboratory and the FDA that finally culminated in their allowing us to administer oral physostigmine.

The advantage of the oral form of the drug, physostigmine, was that it could provide us with a longer acting form. We administered the drug every 2 hours for a number of days to people with Alzheimer's disease.

We hoped through this study to be able to answer the question of whether physostigmine might produce a clinically meaningful change in the lives of people with Alzheimer's, rather than a change on some abstract psychologoical test we were asking them to perform.

Our studies were divided into two phases. First, we find the best doses for each patient, and we administered that dose along the placebo to see if the drug is doing anything. The result of the first 11 patients who received it was that 10 had some improvement.

When we readministered the drug, eight again had an improvement. Unfortunately, in our hands, only three of these people had an improvement that we would call clinically meaningful, which is to say, they were aware of the change, the physician was aware of the change, the nursing staff and their family.

That improvement of approximately 10 percent was hardly as great as one would see, for example, when L-Dopa is given to Parkinsonians or antipsychotic drugs given to schizophrenics.

It was not good enough. However, we knew before we began that physostigmine could not improve the symptoms of every patient with Alzheimer's disease. We knew that for the following reason: For physostigmine to be effective, it has to inhibit the breakdown of acetylcholine.

Since, as this disease progresses, cholinergic cells are dying, there must come a point in which no acetylcholine in substantial amounts is being made, and a drug like physostigmine could not be effective.

The efficacy of physostigmine is dependent on the integrity of the cholinergic neuron. There are other reasons to presume that physostigmine would not be an ultimate treatment for the disease.

Because we knew that it would only be effective in some patients, we set out at the beginning of this study to find a way to predict those people who might receive some benefit from physostigmine.

We found, in collaboration with another colleague in my laboratory, that when we administer drugs that increase brain acetylcholine, the amount of cortisol that people secrete at night goes up, if their brain is normal, and relatively intact, so we reasoned that those people who would have the greatest improvement following physostigmine would be the same people who would have the greatest increment in their nocturnal cortisol.

The people who had the most intact cholinary system would also show an improvement in cognition. Page 7, you see a graph that demonstrates that indeed, that is what happened.

The people who, after receiving physostigmine, had the greatest improvement were those people who had the greatest increase in nocturnal cortisol.

Although these results were encouraging to the scientists for the patient with Alzheimer's disease, the effects were not nearly as impressive or uniform as one would desire. Instead, they only pointed us into other directions.

Nonetheless, these results today with oral physostigmine have now been replicated in five different laboratories, all to about the same degree, the same extent of improvement as we have, although in some cases, a larger percentage of patients have responded in our laboratory.

Where do we go from here? The VA is now considering developing a cooperative study across a number of VA hospitals to identify that subgroup of patients who might respond to cholinergic therapies.

It will not be by any means all patients, and one could argue that the VA is perhaps the worst place to find a population that would respond, because they are generally people with younger disease that is probably more pervasive and affecting more other neurotransmitters.

The strength of the VA is that it has performed cooperative studies before, like drugs for prostate cancer, antipsychotic drugs; and there are the administration and the vehicle in place to perform such work.

Whatever the results of these studies, though we recognize that we will have to do far better with our pharmacological agents, one step toward doing better is to develop animal models, and with the work that Dr. Price modestly reported this morning, we were able to extend some of that, and lesion animals in the area of the brain.

He showed in his slides, projects to the cortex and the hippcampus and has cholinergic cells. By lesioning that area of the brain, we will be able to produce demented animals which don't learn as well, but we have been able to reverse that dementia with a number of cholinergic drugs.

With subsequent funding we received from the National Institute on Aging, we will be extending that model and trying to add other neurotransmitter deficits, and neuromodular deficits to more closely approximate what may be the clinical condition.

I would also want to point out that in doing this kind of research, it is essential that there be a strong collaboration with industry. That has been rather difficult to facilitate.

One company, the Hoechst Roussel Pharmaceutical Co., has taken an interest, and has in fact, based on using this model, some interesting compounds not only reversed the cholinergic deficit, but appeared to increase norepinephrine activity as well.

They are now entering toxicological studies, but they are, in my experience, an exception. Many different kinds of studies must be done to develop effective treatments. We are going to have to conduct other clinical trials with different agents, look to autopsy studies to provide better information, and yet, other preclinical models for our animal studies.

Recent rapid advances in understanding the neurochemistry will lead us, hopefully, to better clinical hypotheses.

To facilitate greater progress, the NIA, with the help of Congress, has designated Alzheimer's disease centers which will help bring us closer to understanding and eventually treating the disease.

I feel compelled to point out that the initial funding levels for these centers is considerably less than can be utilized. To become as effective as they can be, centers will need to see substantial increases in their program over the next few years.

This concludes my statement, Mr. Chairman. I would be pleased to answer any questions that you might have.

[The prepared statement of Dr. Davis follows:]

STATEMENT OF

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and

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before the

U.S. House of Representatives
Committee on Science and Technology
Subcommittee on Investigations and Oversight

September 19, 1984

Mr. Chairman and Members of the Committee: Thank you for the opportunity to speak before you here today. I would like to talk to you about the research that we have been conducting in Alzheimer's disease, a health problem affecting millions of Americans that is well known to this committee.

Alzheimer's disease is now recognized as the most common cause of serious loss of cognitive function in the elderly. The principal symptoms and neuropathologic hallmarks of AD have been known for some time; clinically, these patients are characterized by progressively worsening memory, gradual loss of language ability, and praxis functions, and, occasionally, agitation, violence, or other grossly disturbed behavior. The principal neuropathologic features are the neurofibrillary tangles and senile plaques; because the neuropathologic features are the same regardless of whether onset comes before age 65 (presenile) or after age 65, the term Alzheimer's disease or senile dementia of the Alzheimer type (AD/SDAT) is used to refer to all such patients. Epidemiologic studies indicate that from 5% to 10% of all people older than age 65 suffer various dementias. Of these, approximately 50% have Alzheimer's disease (AD), 20% have multiinfarct dementia (MID), 20%-25% have neuropathologic features of both AD and MID, and the remaining 5%-10% have rarer forms of dementia.

In the United States approximately 1-1.5 million people older than 65 - out of 25 million - probably have AD. Since nearly all victims of AD eventually require complete custodial care, these figures demonstrate both the enormous suffering and personal loss caused by this disease as well as the effect it will have on the cost of health care in our nation.

Although currently there is no treatment for AD that is effective in the majority of cases, recent research on the neurochemistry and psychopharmacology of this disease has raised the possibility that an effective treatment may ultimately be developed.

A series of studies conducted in the United Kingdom in 1976 and 1977 reached an extraordinary conclusion about Alzheimer's disease. Three laboratories working independently reported that in AD, there was a loss of brain cells that made the neurotransmitter acetylcholine. This conclusion was based upon the discovery that the enzyme which synthesizes acetylcholine, and is only found in brain cells that make acetylcholine, (i.e. cholinergic neurons) was very much reduced in the brains of patients with AD.

This finding led a number of research groups, including our own at the Mt. Sinai School of Medicine and the Bronx VA, to suggest that a possible treatment for AD would be a drug that could increase brain cholinergic activity. Such an attempt was particularly appropriate to our research ward, because we had been investigating a drug that increases brain acetylcholine, physostigmine. Physostigmine inhibits the breakdown of acetylcholine. In fact, while neurochemists in the United Kingdom were finding that the brains of patients with AD had a cholinergic deficit, our laboratory was conducting a study of the effects of physostigmine on memory in young normal people. We found that the administration of physostigmine to young normal people could improve their ability to remember new information. The simultaneous discovery of the acetylcholine deficiency in patients with AD, and the ability of drugs that increased brain acetylcholine to enhance memory, led to the idea that the memory disorder in AD might be improved with drugs like physostigmine.

Together, the neurochemical and psychopharmacologic data provided a strong rationale for clinical trials of cholinomimetic drugs in patients with AD. Unfortunately, a limited number of drugs are potential candidates for cholinomimetic therapy. Physostigmine and arecoline, the most readily available cholinesterase inhibitors and cholinergic agonists, respectively, both have the disadvantage of being relatively short acting with half-lives of about 30 minutes. Long acting cholinesterase inhibitors and agonists such as di-isopropyl fluorophosphate and oxotremorine, respectively, are fairly toxic drugs and thus are not good candidates for clinical trials.

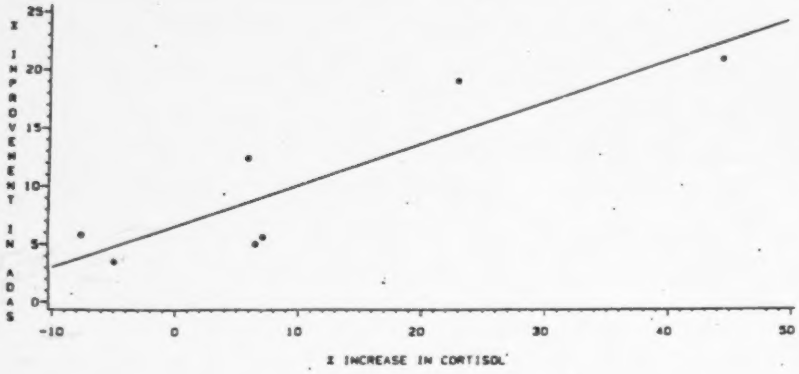
As a result, the earliest trials of cholinomimetic therapy for AD usually involved either choline or phosphatidylcholine (lecithin), precursors to acetylcholine that are safe even in large quantities and which are normally obtained in the diet. It has been demonstrated in rats that increases in dietary choline or phosphatidylcholine are followed by increases in the concentration of brain acetylcholine. Clinical trials, however, failed to demonstrate any acute effect of these precursors on memory or other aspects of cognition, either in unaffected people or in patients with AD. The reason for this failure appears to be that precursors do not substantially affect the rate of cholinergic transmission even though they may increase acetylcholine concentrations.

With the help of a program project grant from the National Institute of Aging, as well as support from the Veterans Administration, my colleague Richard Mohs and I began a study of physostigmine in AD. We first administered an intravenous form of the drug and then an oral preparation. The initial studies with intravenous physostigmine were encouraging, although the ability of physostigmine to improve memory was hardly as robust as one might have hoped. Still, additional studies were necessary, because intravenous physostigmine is so short acting that it was impossible to adequately assess whether the drug would help patients in daily life.

Studies with oral physostigmine have begun. The drug is given every two hours from 7 a.m. to 9 p.m. in doses of 0.0, 0.5, 1.0, 1.5 and 2.0 mg each for three to five days. On the last day, the patients' overall symptoms are rated with the Alzheimer's Disease Assessment Scale (ADAS) at each dose. This instrument, developed in our laboratory, measures all of the principal symptoms associated with AD including memory loss, dysphasia, dyspraxia, agitation, and mood disturbance. The dose of oral physostigmine associated with the least severe symptoms is then randomly readministered along with placebo for three to five days under double-blind conditions.

Of the first 11 patients who completed this study, 10 showed some decrease in symptoms with at least one of the physostigmine doses. Of those, 8 showed less severe symptoms while receiving physostigmine in the replication phase of the study than they did while receiving placebo. From a clinical perspective, only 3 of the patients demonstrated enough improvement to be clinically relevant. On the ADAS these patients showed at least a 10% improvement over placebo. However, there is very good reason to believe that not all patients with AD will respond to this drug, nor will all patients who respond to physostigmine respond in the same way. This problem arises from the fact that for physostigmine, which prevents the breakdown of existing acetylcholine, to work, the brain must be able to synthesize some acetylcholine. Unfortunately, as the disease progresses cells which synthesize acetylcholine are dying. Because of this problem we needed some way, besides memory testing, to be sure the physostigmine was getting into the patients' brains. In collaboration with an endocrinologist in our laboratory, Dr. Bonnie Davis, we have found that when physostigmine enters the brain, and acetylcholine containing cells are available to synthesize adequate quantities of acetylcholine, the physostigmine increases the amount of cortisol the patient's body secretes at night. The effect of physostigmine to increase nocturnal cortisol concentrations correlates with the drug's ability to improve the symptoms of AD, as the figure below indicates. These results support the notion that physostigmine only improves the cognitive functioning in those patients whose acetylcholine containing neurons have not yet been devastated by the disease.

CORTISOL vs COGNITIVE
RESPONSE TO ORAL PHYSOSTIGMINE



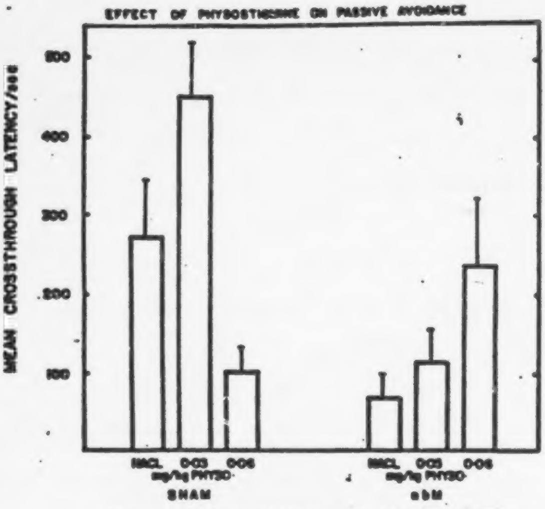
$r=.88 \quad p < .01$

Although these results are encouraging to the scientist, for the patient with AD the effects are not as impressive or uniform as one would desire. Instead, they only point to other directions that must be pursued and better drugs that must be developed. Nontheless, these results with oral physostigmine, which have been replicated in at least 5 other laboratories, indicate that current approaches are rational.

Given these effects of physostigmine, as well as results from other researchers, there now is a need to identify those Alzheimer's patients who may be made modestly less demented by therapies that increase brain acetylcholine. A cooperative study has been proposed by Richard Mohs and myself, as well as medical scientists from a number of other VAs, to determine the ultimate clinical utility of physostigmine. We are hopeful that it will be funded, and over the next five years, may be able to ascertain just what segment of the Alzheimer's population may be benefited by physostigmine. Such a study can only be done through the cooperative studies program at the Veterans Administration. It will require extensive coordination of the large study population that is made available by the health care delivery system in the Veterans Administration. The cooperative studies program has produced invaluable studies of the effects of treatment of high blood pressure, and prostate cancer, yielding information which has saved lives, prevented disability, and saved health care dollars.

Whatever the results of the studies in progress, it is clear that not all patients are going to be helped by physostigmine treatment, and that those patients who are helped will not be returned to a normal life. For that reason, additional work is necessary. To that end, our program has been engaged in developing an "animal model" of Alzheimer's disease that may expedite development of other drugs. These studies are made possible by a merit review grant from the VA to me and my colleague, Dr. V. Haroutunian, as well as collaboration with the Hoechst Roussel Pharmaceutical Corporation. Together, we have found that destruction of a tiny part of the rat's brain can produce a deficit in acetylcholine that in many ways resembles the deficit that exists in AD. This rat model makes possible the screening of drugs for their ability to improve the memory of "demented" animals.

The figure below illustrates how physostigmine, the drug we are administering to humans, can reverse the abnormal behavior that animals with a cholinergic deficit exhibit. Other drugs such as oxotremorine and 4-aminopydirine, are equally effective at normalizing these animals.

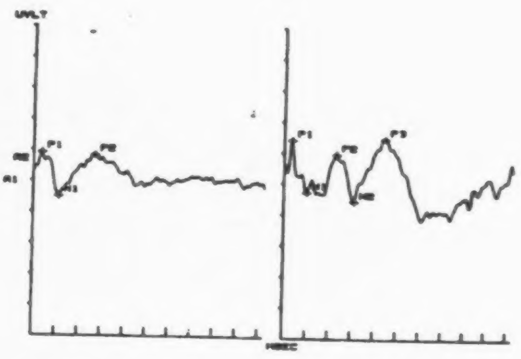


Effects of post-acquisition physostigmine administration on the 72 hour retention of passive avoidance in nbm lesioned and sham operated rats.

In the future, and with the help of the NIA's new Alzheimer's Disease Research Center, new drugs that might be more efficacious will be tested, and more importantly, the model will be extended so that it even more closely resembles the human condition. Other neurotransmitter and neuromodular deficits, particularly of noradrenergic neurons and somatostatin concentrations, will be induced. In addition, with the help of funding from the VA and the NIA, we are now transplanting brain tissue to these lesioned animals in the hope of normalizing their behavior. This will be a difficult process, but one with obvious implications for developing a treatment for Alzheimer's disease.

A real problem in AD research is the difficulty in making this diagnosis with absolute certainty. At present, a definitive diagnosis of AD is only made at autopsy by examining the brain under the microscope, or with the help of a biopsy. Rarely are biopsies performed, thus some uncertainty inevitably exists when a clinical diagnosis is made. For that reason our program, with the help of funding from the NIA and the VA,has been searching for a clinical test for AD that would facilitate a diagnosis prior to autopsy.

The figure below is a representation of electrical activity recorded from the scalp when normal people hear a series of similar tones (left panel). These wave forms can be generated and recorded with uncanny reliability. However, as the panel on the right indicates, when similar tones are interrupted by a different tone, additional waves appear.



The table below illustrates that patients with AD show a very different response to those tones than normal elderly.

Discriminant Function Coefficient	
P2 Latency to "Frequents"	-0.41
N2 Latency to "Rares"	0.62
P3 Latency to "Rares"	0.49
P3 Amplitude to "Rares"	-0.43

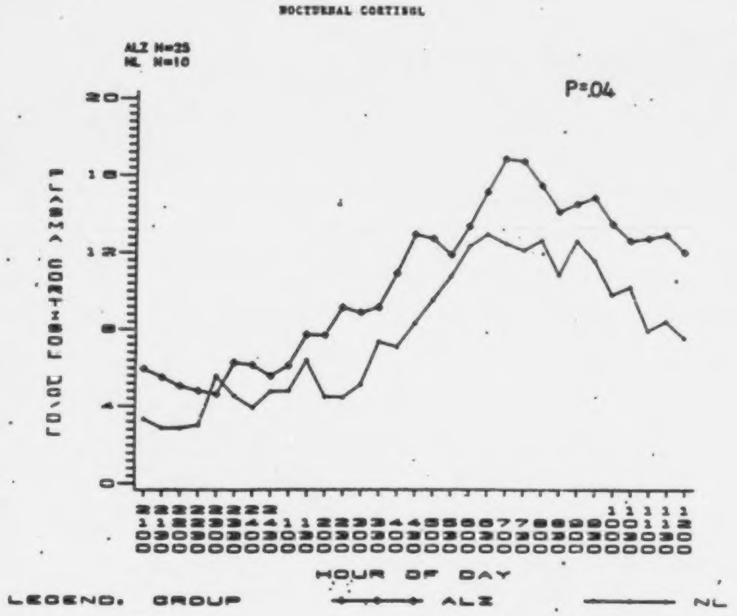
Actual Group	N	Predicted Membership	
		Correct:	Incorrect:
Alzheimer's	12	12	0
Controls	16	15	1

Percent of "grouped" cases correctly identified: 96.4%

We have so far been very accurate in using these measurements of electrical activity to classify patients as having AD or being without dementia. This is not the same as saying that this technique can differentiate Alzheimer's patients from patients with other forms of dementia, but it is at least an encouraging first step. An answer to that question will be provided by work that will be conducted by our Alzheimer's Disease Research Center. The principal investigator in these studies is Dr. Thomas Horvath.

The data that he has so far generated encourages us to look at scalp electrical activity in far more depth to determine if it might be a diagnostic marker of Alzheimer's disease.

Another possible marker is concentrations of plasma cortisol in blood. The pattern of nocturnal cortisol secretion, as shown in the figure below is higher in patients with AD than normal elderly people of roughly the same age. Other aspects of cortisol



secretion seem equally unusual. For example, cortisol in a single sample of blood taken at 9 a.m. was, in fifteen or sixteen instances, higher in Alzheimer's patients than in age and sex matched normal persons. These abnormalities also suggest that measurements of cortisol may help differentiate Alzheimer's from normal people, and perhaps other forms of dementia. However, it is important to emphasize that these are initial studies and require much more work before we can take them as definitive.

Clearly, many different kinds of studies must be done in order to develop effective treatments and better diagnostic techniques for AD. Some of these will involve clinical trials of new treatments or diagnostic tests, others will be autopsy studies, and still others will be preclinical studies of animal models. Recent rapid advances in understanding the neurochemistry of this disease have provided a rational, scientific basis for designing both clinical and preclinical research programs. In order to facilitate even greater progress, the NIA, with the help of Congress, has designated Alzheimer's Disease Centers. I have the good fortune to direct one of the approved centers at the Mt. Sinai School of Medicine. Hopefully these centers will bring us closer to understanding, and eventually treating this disease. However, the initial funding levels for these centers is considerably less than the amount that could be utilized. To become as effective as they can be, centers will need to [illegible] a substantial increase in this program over the next few years.

This concludes my statement. I would be pleased to answer any questions that you or members of the Committee may have.

Mr. GORE. Thank you very much. My questions are not going to be as extensive as they might be if we hadn't gone longer than anticipated today.

You all have been very patient. Let me ask a few questions, and we will follow up with some questions in writing about some of the more technical aspects of your testimony.

Dr. Wells, how do you distinguish between the depression that is not uncommon among elderly patients, and the depression that is an early sign of Alzheimer's?

Dr. WELLS. First of all, I often can't—and which would be the way I suppose I should begin, but usually, when we encounter a depression which is, should be looked upon as a disease, if you like, or a clinical disorder, it comes on rather quickly.

There is a rather rapid downhill course and in the elderly, in particular, it may be marked a great deal by physical symptoms and also by loss of memory and loss of ability to concentrate, many of the early signs of Alzheimer's disease, in other words.

So early in the course, it may be very difficult to distinguish these two disorders, which is a major reason why in those instances it may be very valuable to go ahead and treat for depression, but first of all, you may help it, and secondly, you may be missing a diagnosis if you don't go ahead in that direction.

Mr. GORE. Are patient visits to your office covered by insurance?

Dr. WELLS. In many instances they are. But Medicare is particularly poor in covering psychiatric care for the elderly, both on an outpatient or an inpatient basis, which means that for treatment of many of the behavioral problems associated with Alzheimer's disease, which perhaps could be cared for on an outpatient basis, there is almost no funding available through Medicare.

Mr. GORE. From your perspective, what do you think are further areas in which research is needed on behavioral aspects of the disease?

Dr. WELLS. I think really most of what we know about the ablity to care for behavioral aspects in Alzheimer's disease remains at the anecdotal level. That is that very few hard studies have been done about just what is the most effective way to treat them, with a variety of treatment methods available.

It is still in many ways using one's experience to predict what is going to happen rather than having really solid studies which can be generally applicable to physicians across-the-board.

Mr. GORE. I see.

Dr. Price, have the experiments you described on animal models been on monkeys only?

Dr. PRICE. No. I think that, as Ken described, some of those studies are done on rodents. And I think for the most part those kinds of studies should be done on rodents, where you can have large numbers of animals. However, when you are looking at higher order behaviors—Ken might disagree, but rodents, I don't think, have that high order of behavior, while nonhuman primates do, and their brains are very similar to humans.

So I think for very selected questions, nonhuman primates are probably more useful. But all of that approach should be screened first through rodents.

Mr. GORE. Now, on page 4 of your testimony you mentioned as one of the therapeutic approaches providing precursors such as choline and lethicin. A recent article in a magazine warns against cure-alls, and specifically mentions lecithin as ineffective.

Could you comment on that?

Dr. PRICE. I think Ken alluded to that. I think that a combination of choline and lecithin really is not effective in terms of improving activities of daily living. And I certainly would not recommend that as a sole treatment to anyone.

Mr. GORE. All right. So you would not recommend lecithin.

Could you explain the sentence in the last paragraph of your testimony, "The development of new therapeutic strategies for Alzheimer's disease will be enhanced by further knowledge of transmitter populations at risk."

Dr. PRICE. It is a little jargoney, isn't it? I think basically one of the things we need to know, certainly not the only thing, about Alzheimer's disease and many of these other degenerative disorders are what are the characters of these cells.

I mean what transmitters do they use, how do they act in the normal brain, and then what happens to them in disease. I think many of these cell populations at risk we have relatively little knowledge about what they do.

We are really talking about anatomy, physiology, chemistry of normal systems. And I think that kind of information, though it is not directly relevant to Alzheimer's disease, is, in fact, the substrate upon which people like myself will be able to do disease-related research.

For example, the question that you had earlier with regard to Bill Field's work, basically in Parkinson's disease you have a loss of neurons that use dopamine in the brain and the patients are stiff and don't move very rapidly.

In rodents, investigators have destroyed that subpopulation and have transplanted embryonic dopaminergic nerve cells into target sites, those nerve cells take, they re-innervate the target site and the rodents do better. Actually, Bill Field and Ed Wyant at NIMH are doing those kinds of studies now in monkeys.

I will go back this afternoon and look at some slides they have sent me.

Finally, there have been—I don't like to use the word "transplants," but there have been grafts placed in human patients. In Sweden, Lars Olsen and coworkers have put adrenal gland transplants into the target cell population of two Parkinsonian patients. We don't know whether those transplanted cells have taken or been of any benefit.

Theoretically those kinds of issues could be looked at even in living patients with PET scans.

Mr. GORE. Dr. Davis, you mentioned brain cell transplants on page 9 of your testimony. How far along is this experiment? How practical would it be if applied to Alzheimer's patients at some point in the future?

Dr. DAVIS. We were visited about 2 years ago by Anders Bjurkland, who has really established some of the pioneering work in this area. And together he and I, and also we have talked to Don about this, are engaged in a program in which we are looking at very different aspects of the problem.

In our work so far we have taken embryonic tissue, we have placed it in the hippocampus, and we have so far been able to see innervation and some normalization of behavior. That really is no more than has been done by Bjurkland.

We are looking toward, as I know our other labs, and seeing whether we cannot get some of the deficit in the basalis to the cortex to be reversed by some grafting techniques. That is going to be a much, much more difficult problem.

I would say we have enormous scientific leaps that must be made in the laboratory before this could be of any use to patients. But there are a lot of convergent pieces of evidence including growth factors that may help these transplants or grafts take that can lead us to some optimism in the long run.

Mr. Gore. Let me say that we have a vote on the floor. We may follow up with some written questions.

I want to thank you for your testimony here today. You have been most helpful.

I want to thank all of the witnesses today.

Tomorrow we have a very interesting day which will begin at 9:30 in this room, and we will hear from the two researchers that I talked about earlier, Dr. Marotta and Dr. Davies. We will also hear about other possible causes of the disease, and then we will have a final panel on care and financing which will be particularly relevant in the light of the expected release by Secretary Heckler of their long-term study of this matter tomorrow, and we will look forward to tomorrow's testimony.

Thank you all. We will adjourn until tomorrow morning.

[Whereupon, at 1:25 p.m., the subcommittee adjourned, to be reconvened Thursday, September 20, 1984.]

ALZHEIMER'S DISEASE RESEARCH

THURSDAY, SEPTEMBER 20, 1984

HOUSE OF REPRESENTATIVES,
COMMITTEE ON SCIENCE AND TECHNOLOGY,
SUBCOMMITTEE ON INVESTIGATIONS AND OVERSIGHT,
Washington, DC.

The subcommittee met, pursuant to adjournment, at 9:38 a.m., in room 2325, Rayburn House Office Building, Hon. Albert Gore (chairman of the subcommittee) presiding.

Present: Representatives Gore, Skeen, Winn, and Schneider.

Staff present: James C. Greene, science consultant; Robert W. Lamina, minority technical consultant.

Mr. GORE. The subcommittee will come to order. Let me welcome all of our witnesses and guests. I am delighted that you are all here. This is the third in a series of hearings that we have been holding on Alzheimer's disease. Today, we will conclude our hearings, and we will begin the process of compiling all of the testimony and evidence that has been presented to us, and then we will have a comprehensive report with recommendations on how the Congress ought to proceed in lifting the priority assigned to our national effort to deal with Alzheimer's disease, to accelerate our efforts to research its causes and cures, and to improve our efforts to help patients and families who have to cope with this disease.

The first day of these hearings, held in Johnson City, TN, concentrated on health care issues. Yesterday, we heard testimony from caregivers, from representatives of Federal agencies currently funding research on Alzheimer's disease, and on scientists. The caregivers, spouses of victims of Alzheimer's disease, described the physical, financial, and emotional suffering borne by family members and their heroic efforts to cope, despite the lack of resources and the lack of alternatives. In Alzheimer's disease, as one witness said, Mrs. Grashot, there are always two victims: the patient and the caregiver.

The Federal representatives from the Public Health Service discussed the types of research funded by the agency in this area and commended the Congress for focusing more attention on diseases that are chronically debilitating. After listening to the testimony of several leading research scientists, it was apparent that exciting scientific findings have indeed developed within the last 10 years, allowing a better understanding of the disease. These very findings have then raised new questions which are opening the pathways for further and more coordinated research. We are hopeful that we are beginning to see the light at the end of the tunnel.

Today, we will hear testimony from scientists, who will discuss several research programs investigating possible causes of Alzheimer's disease. In addition, witnesses will testify about home care for Alzheimer's disease victims and about the various options in health care financing.

I am very pleased that our first two witnesses are among the leaders in making new discoveries in this area. I referred in my prepared statement here to the breakthroughs over the last 10 years. We have had a couple of exciting breakthroughs reported in the last few weeks, and we are going to look forward to hearing about those this morning.

I want to now recognize my distinguished colleague and friend, the ranking minority member of this subcommittee, Mr. Skeen.

Mr. SKEEN. Thank you, Mr. Chairman. I have no prepared statement, other than to welcome the witnesses. I understand yesterday's hearing went very well, and I want to commend you for holding these hearings. With that, I will relinquish my time back to you, Mr. Chairman, and we will get on with the hearings.

Mr. GORE. It is a pleasure to work together, and let me also ask unanimous consent to hold the record open at this point for inclusion of a statement by Congressman Larry Winn, the ranking Republican member of the full committee who has played such an important role in designing these hearings. And also I would like to ask unanimous consent to include a statement from the Honorable Bill Lowery of California, who is a member of the full committee and has a statement on this subject, and such other members of the subcommittee who may care to put their remarks in the record.

Without any further ado, let's go to our first panel of witnesses: Dr. Charles Marotta, from the Laboratory of Psychiatric Research at McLean Hospital, in Belmont, MA, and Dr. Peter Davies, associate professor of pathology and neuroscience at the Albert Einstein College of Medicine in the Bronx. We are delighted to have both of you here.

There are in the audience today, I might tell you, several people who are members of families of patients with Alzheimer's disease, either presently or formerly. If I may take the liberty of speaking for them for just a moment, I want to thank you two distinguished scientists for devoting so much of your lives to trying to find a cure for this terrible illness. When our society is afflicted with something this horrible, we turn to the brightest minds we have, and we just hope and pray that there will be those who are dedicated enough and smart enough to get in there and dig for the answers. We know we are a long way from the solution to this puzzle, but you two, just in the last few weeks, have excited the scientific community with some real breakthroughs.

We are delighted to have you together here in public. I do not know if it is the first time, but it is the first time since the breakthroughs took place, and we are just really glad you are here. Without objection, your complete prepared statements will be included in full in the record, and you are encouraged to proceed in any way you feel most comfortable. If you want to make a formal presentation by reading, that is fine. If you want to summarize it, that is fine. But I would ask if it is at all possible to just try to make it understandable to those of us who have not had your years of training and do not have your intellect either, for that matter. Please do your best to describe it in terms that we can follow.

Dr. Marotta, we will begin with you and then go to Dr. Davies. Welcome, and please proceed.

STATEMENT OF CHARLES A. MAROTTA, M.D., PH. D., LABORATORY OF PSYCHIATRIC RESEARCH, McLEAN HOSPITAL, BELMONT, MA

Dr. **Marotta**. I am Charles Marotta. I am associate professor of neuroscience in psychiatry, Harvard Medical School, and I am chief of the Molecular Neurobiology Laboratory at McLean Hospital, which is a Harvard teaching hospital. I do have a prepared statement. It is brief.

Alzheimer's disease is a chronic disorder of the brain that occurs in the later decades of life and leads to dementia, involving intellectual deterioration with deficits in memory and reasoning. Alzheimer's disease is characterized in brain by hallmark lesions of neurofibrillary tangles, neuritic plaques, and a loss of neurons. I think these have been discussed by others.

Mr. **Gore**. We understand that because we saw slides yesterday. The neurofibrillary tangles are little bundles of nerves——

Dr. **Marotta**. They are fibers.

Mr. **Gore**. Fibers that presumably block the normal functioning of the cells where they are found.

Dr. **Marotta**. Right. Notably, there are declines in certain enzymes of neurotransmitter metabolism, and Dr. Davies will be addressing that in some detail.

Observations made by medical scientists on Alzheimer's disease have followed a classical route. The postmortem brains of patients who had dementia in life were examined and were shown to contain an abundance of both plaques and tangles. Thus, there was a change in the morphology or structure of certain brain regions. Research on Alzheimer's disease was then moved forward a step when scientists began to ask whether or not an altered function, in addition to altered structures, may be involved. In fact, this has been shown to be true, most notably with the work of Dr. Davies on the cholinergic neurotransmitter system. These observations were then advanced by the scientists at Johns Hopkins University, who presented evidence that the subcortical neurons in the brain that are responsible for the major cholinergic innervation of the cortex are themselves undergoing degeneration in Alzheimer's disease.

Mr. **Gore**. I was just thinking that.

Dr. **Marotta**. Memory deficits appear to be associated with these cholinergic enzymes and the cholinergic neurons, and this work was recently extended by the studies of Hyman and colleagues who have shown that the hippocampus, a part of the brain also associated with memory, appears to have lost major inputs and outputs in Alzheimer's disease. My point is that anatomic observations and pharmacologic studies have given us a useful picture of certain kinds of changes in the demented brain.

If I understand the purpose of today's hearing, it is to ask, essentially, where we ought to be going in future studies on Alzheimer's disease. There are a number of answers to this question, and the witnesses who address the committee will have different suggestions, I expect. My own answer is based upon my particular interest. I believe that the field of Alzheimer's research would benefit most from a movement toward more basic science investigations that focus on molecular biology. Since we know that in dementia certain neurons degenerate and certain enzymes are less effective, we must ask a simple question, which is: By what fundamental processes are these abnormal events triggered in the first instance.

The question appears simple but is broad in its scope since it relies upon the fundamental elements of cellular life, which include the structure and function of DNA, ribonucleic acid [RNA], and proteins. A simple overview of the processes we are concerned with would be the following facts: the DNA in the nucleus of the cell is responsible for producing RNA, which in turn is responsible for synthesizing the proteins in the body of the cell. The proteins, including enzymes, that are made are responsible for maintaining the orderly functioning and the survival of neurons and cells of all types. In this description of the life of the cell, I have omitted the numerous intervening steps in the interest of time. I can list a few thousand for you, if you wish.

Mr. **Gore**. That is OK.

Dr. **Marotta**. It seems to me then that it is unlikely that the extensive cellular pathology that we know exists in the brains of patients with Alzheimer's disease can occur by mechanisms that are entirely independent of DNA and RNA, since the latter control the normal functioning of the cell.

I would like to suggest that fruitful new research on Alzheimer's disease could be realized if basic scientists from other disciplines were lured into dementia research and, in addition, if there were incentives to investigators already committed to Alzheimer's disease to move to more molecularly oriented projects.

If these suggestions appear somewhat obvious, you may be asking yourselves why it is that basic scientists have largely ignored research on dementia and on the human brain in general. There are several answers. The first is that because of ethical considerations, the medical community does not remove brain specimens by biopsy from living patients for purely research purposes. The second reason is that there are no experimental animal models that exactly mimic the pathologic changes found in the human brain in Alzheimer's disease. Thus, it seemed for some time, there was no suitable tissue to work with that could effectively be used for basic molecular research.

Several years ago, I suggested that we attempt to use donated frozen postmortem brains for molecular biological studies. There are several brain banks in this country whose mission is to accept donated brains and to store frozen specimens that are then made available to investigators. This source of material has worked well in the past for a number of enzyme studies. The postmortem tissue was not, however, used for macromolecular studies, since it had been assumed that DNA and RNA were too fragile to survive the postmortem interval prior to freezing because of the body's own degradative processes. The postmortem interval is typically a matter of hours, and samples are stored frozen sometimes for years before they are used for research.

We have shown, however, that frozen and stored postmortem brain can, in a number of cases, be used to prepare RNA that retains some of its original functional activity. That is, a fraction of the postmortem RNA can be extracted and, under the proper laboratory conditions, be used to synthesize human brain proteins that were made in life. The proper experimental conditions are standard ones that have been developed in a number of laboratories over the years. We adapted them for use with the autopsied brain material. Our original observation that human postmortem RNA remains sufficiently functional for experimentation has now been verified in at least four other laboratories on an international scale.

We applied these methods to the donated and frozen brains of Alzheimer's disease victims that were given to us by Dr. Davies and Dr. Terry at Einstein. We observed that the most demented brain areas had lower amounts of extractable RNA, which was less active in producing new protein. We found that one of the contributing factors was the excessively high activity of the brain enzyme that destroys RNA. We suggested that this observation might indeed be a facet of the disease. This suggestion was subsequently confirmed to some extent by French scientists who use the noninvasive technique of positron emission tomography, or PET scanning, as their basic tool. These scientists demonstrated that the brains of living patients who are severely demented produce only about half the amount of protein that is normally made in nondemented individuals of the same age.

Our biochemical findings are consistent with the PET scanning studies. However, they also ask more questions than they answer. Among these are: What is the exact relationship between our findings concerning brain RNA and protein synthesis and the classical features of Alzheimer's disease. Are our findings related to the loss of nerve cells and neurotransmitter enzymes?

In terms of future research directions, I believe that ultimately the most useful outcome of our present research efforts is that since human brain polynucleotides have been shown to have survived in the postmortem state, they can be used for recombinant DNA experiments. By means of these techniques, we may see—and I expect we will see—an explosion of new research endeavors that are directed toward elucidating the fundamental nature of neuropsychiatric diseases.

These questions bring me to the last part of my presentation, which concerns how best to carry out research that is of a complex, costly, and time-consuming nature. Fundamental molecular investigations require a stable research environment. The typical 3-year period for competitive grants is often long enough to carry out only the preliminary basic research studies. A longer period of time is often needed to perfect new procedures and to demonstrate the usefulness of newer technology. Investigators are quite aware that support may be lost at the time they become most proficient in their areas of expertise. This situation contributes to the anxiety of research and affects performance. The same kind of problems arise with respect to longer 5-year program grants, due to the more comprehensive scope of their activities.

An extraordinary amount of time is consumed by investigators as they prepare grant requests. Over the last several years, a substantial portion of my time has been devoted to preparing new grant applications and renewing previous applications when that time really should have been spent in the laboratory.

Fundamental research often moves slowly and relatively few scientific publications may be produced in the time period designated by the National Institutes of Health for project and program grants. These comments apply in particular to studies of the central nervous system, where progress is slow due to the complex nature of the subject. As I have stated, neuroscientists have only recently borrowed extensively from cellular and molecular biology and are still learning to apply these tools to the central nervous system. In one sense, the neurosciences are among the oldest areas of biology, and in another sense they are among the newest. Fundamental research becomes even more tedious and complex when we try to understand abnormal neuronal functioning as in Alzheimer's disease. Thus, the need for long-range goals is, in part, dictated by the system under investigation.

A substantial increase in funding for molecular biological studies of aging and dementia at this time appears to me to be a brilliant investment that will almost certainly have a huge payoff in the future, both in terms of alleviating the suffering of victims of Alzheimer's disease and in terms of decreasing the financial burden of providing chronic medical and nursing care for our afflicted elderly population.

Thank you.

[The prepared statement of Dr. Marotta follows:]

Statement to: The Subcommittee on Investigations and Oversight of the Science and Technology Committee

September 20, 1984

Charles A. Marotta, M.D., Ph.D.
Harvard Medical School and McLean Hospital

Alzheimer's disease (AD) is characterized in brain by the hallmark lesions of neurofibrillary tangles, neuritic plaques and a loss of neurons; in addition, there is a decline in certain enzymes of neurotransmitter metabolism.

Observations made by medical scientists on AD have followed a classical route. Brains of patients who had dementia in life, were examined and shown to contain an abundance of plaques and tangles. Thus, there was a change in the morphology or structure of certain brain regions. Research on AD was then moved forward a step when scientists began to ask whether or not an altered function, in addition to altered structures, may be involved. And, in fact this has been shown to be true, most notably with the work of Dr. Peter Davies on the cholinergic neurotransmitter system. These observations were then advanced by the scientists at Johns Hopkins University who presented evidence that the subcortical neurons in the brain that are responsible for the major cholinergic innervation of the cortex are themselves undergoing degeneration in AD. Memory deficits appear to be associated with the cholinergic enzymes and the cholinergic neurons; and, this work was recently extended by the studies of Hyman and colleagues who have shown that the hippocampus, a part of the brain also associated with memory, appears to have lost major inputs and outputs in AD. Thus anatomic observations and pharmacologic studies have given us a useful picture of certain kinds of damage in the demented brain.

If I understand the purpose of today's hearing, it is to ask, essentially, where we ought to be going in future studies on AD. There are a number of answers to this question and the witnesses who address the committee will have different suggestions. My own answer is based upon my particular interests. I believe that the field of AD research would benefit most from a movement toward more basic science investigations that focus on molecular and cellular biology. Since we know that in dementia certain neurons degenerate and certain enzymes are less effective, we must ask a simple question: viz., by what fundamental processes are these abnormal events triggered in the first instance.

The question appears simple but is broad in its scope since it relies upon knowledge of the fundamental elements of cellular life which include the structure and function of DNA, ribonucleic acid (or RNA), and proteins. A simple overview of the processes we are concerned with would be the following facts: the DNA in the nucleus of the cell is responsible for producing RNA which in turn is responsible for synthesizing proteins in the body of the cell. The proteins, including enzymes, that are made are responsible for maintaining the orderly functioning and the survival of neurons and cells of all types. In this description of the life of the cell I have omitted several thousand intervening steps in the interest of time. These include complex and inter-related regulatory mechanisms that act at the level of DNA, RNA, protein synthesis, protein modification, protein functioning and, finally, at the level of degradation. Thus, it seems to me that it is unlikely that the extensive cellular pathology that we know exists in the brains of patients with AD can occur by mechanisms that are entirely independent of DNA and RNA since the latter control the normal functioning of the cell.

I would like to suggest that fruitful new research on AD could be realized if basic scientists from other disciplines were lured into dementia research; and, in addition, if there were incentives to investigators already committed to AD to move to more molecularly oriented research projects.

If these suggestions appear somewhat obvious you may be asking yourselves why basic scientists have largely ignored research on dementia, and on the human brain in general. There are several answers. The first is that because of ethical considerations the medical community does not remove brain specimens by biopsy from living patients for purely research purposes. The second reason is that there are no experimental animal models that exactly mimic the pathologic changes found in the human brain in AD. Thus, it seemed, for some time that there was no suitable tissue to work with that could effectively be used for basic molecular research.

Several years ago I suggested that we attempt to use donated frozen postmortem brains for molecular biological studies. There are several brain banks in this country whose mission is to accept donated brains and to store frozen specimens that are then made available to investigators. This source of material has worked well in the past for a number of enzyme studies. The postmortem tissue was not, however, used for macromolecular studies since it had been assumed that DNA and RNA were too fragile to survive the postmortem interval prior to freezing due to the body's own degradation processes. The postmortem interval is typically a matter of hours and samples are stored frozen, sometimes for years before they are used for research.

We have shown, however, that frozen and stored postmortem brain can, in a number of cases, be used to prepare RNA that retains some of its original functional activity. That is, a fraction of the postmortem RNA can be extracted and, under the proper laboratory conditions, be used to synthesize human brain proteins that were made in life. The proper experimental conditions are standard ones in molecular research that have been developed over the years. We adapted them for use with the autopsied brain material. Our original observation that human postmortem RNA remains sufficiently functional for experimentation has now been verified in at least four other laboratories on an international scale.

We applied these methods to the donated and frozen brains of AD victims that were obtained from Dr. Davies and Dr. Robert Terry at Albert Einstein College of Medicine. We observed that the most demented brain areas had lower amounts of extractable RNA which was less active in producing new protein. We found that one of the contributing factors was the excessively high activity of a brain enzyme that destroys RNA. We suggested that this observation might be a facet of the disease. This suggestion was subsequently confirmed by French scientists who use the non-invasive technique of positron emission tomography or PET scanning as their basic tool. These scientists demonstrated that the brains of living patients who are severely demented produce only about half the amount of protein that is normally made in non-demented individuals of the same age.

Our biochemical findings are consistent with the PET scanning studies, however they also ask more questions than they answer. Among these are: What is the exact relationship between our findings concerning brain RNA and protein synthesis and the classical features of AD? Are our findings related to the loss of nerve cells and neurotransmitter enzymes?

These questions bring me to the last part of my presentation which concerns how best to carry out research that is of a complex, costly and time-consuming nature.

Fundamental molecular investigations require a stable research environment. The typical three year period for competitive grants is barely long enough to carry out more than preliminary basic research studies. A longer period of time is often needed to perfect new procedures and to demonstrate the applicability of newer methodology. Investigators are quite aware that support may be lost at the time they become most proficient in their areas of expertise. This situation contributes to the anxiety of research and affects performance. The same kind of arguments can be applied to existing program grants due to the more comprehensive scope of their activities. An extraordinary amount of time is consumed by investigators as they prepare grant requests. My laboratory and staff are supported entirely by grants; I would estimate that over the last two years 30% to 40% of my time has been devoted to preparing new grant applications.

Fundamental research often moves slowly and relatively few scientific publications may be produced in the time period designated by the National Institutes of Health for project and program grants, i.e., 3 or 5 years. These comments apply, in particular, to studies of the central nervous system where progress is slow due to the complex nature of the subject. As I've stated, neuroscientists have only recently borrowed extensively from cellular and molecular biology and are still learning to apply these tools to the CNS. In one sense the neurosciences are among the oldest areas of biology, and in another sense they are among the newest. Fundamental research becomes even more tedious and complex when we try to understand abnormal neuronal functioning, as in Alzheimer's disease. Thus, the need for long-range goals is, in part, dictated by the system under investigation.

I would like to close by offering several suggestions.

1. Basic molecular and cell biological research on AD should be a top funding priority of the Congress.

2. In this targeted area of research, there should be an opportunity to routinely apply for a 5-year grant outside of the Program Project mechanism.

3. Program grants should be extended to 7 years or longer.

4. The budget of the National Institutes of Health for research on human aging and Alzheimer's disease should be substantially increased.

A substantial increase in funding for aging and dementia research at this time appears to me to be a brilliant investment that will almost certainly have a huge payoff in the future - both in terms of alleviating the suffering of victims of AD and in terms of the financial burden of the country.

Mr. GORE. Thank you very much. We will certainly take note of these recommendations. We have several questions, but we will hear from Dr. Davies first.

Dr. Davies, we are delighted that you are here, and we invite you to proceed.

STATEMENT OF DR. PETER DAVIES, ASSOCIATE PROFESSOR OF PATHOLOGY AND NEUROSCIENCE, ALBERT EINSTEIN COLLEGE OF MEDICINE, BRONX, NY

Dr. DAVIES. I will not read at least the first half of my prepared statement. I would really like to tell you my perception of how research on Alzheimer's disease has progressed. It is important to recognize the two stages of research on Alzheimer's disease. The first stage is looking at the symptoms of the patient and asking what causes the symptoms of the disease, like looking at a patient with a headache and saying, "This patient has a headache. What is the cause of the headache?" Then we can treat symptoms. That stage of the research on Alzheimer's disease, I believe, is coming to something of a close.

The very extensive neurochemical studies that have gone on over the last 8 years on Alzheimer's disease have revealed a very specific deficit in the concentration of a neurotransmitter. This is really just a chemical in brain which is used for communication. The transmitter is acetylcholine, and you have seen a lot in the press about neurochemical deficits causing Alzheimer's disease. This is inaccurate, because the neurochemical deficits cause the symptoms of Alzheimer's disease. We have, I think, now very good evidence that at least some of the major symptoms of Alzheimer's disease are caused by a deficiency of acetylcholine. That is pure and simple.

There are, in some cases of Alzheimer's disease, other chemical deficits. That is true. There are three which I would bring up: somatostatin; another neurotransmitter, substance P; and noradrenalin. These deficits in these chemicals are found. They are not as consistently found as the deficit in acetylcholine, nor does our vast stock of basic research suggest that these deficits cause the symptoms of Alzheimer's disease. So our first goal then is to try and identify what is producing the immediate symptoms of the patient, and I think we have done that. This is a deficiency of acetylcholine.

Just this year, in the last year-and-a-half, we have seen the first statistically significant improvements in patients. These have resulted from attempts to treat the acetylcholine deficiency directly. It must be stressed that these improvements in patients are statistically but not clinically important. These are reproducible improvements in patients which do not make a great deal of difference to the life of the patient or the family, but it is a first step. It is the first time that this has been accomplished.

Further research with the pharmaceutical industry involved to develop better treatments for acetylcholine deficiency is essential. Even if other deficiencies are found in the Alzheimer brain consistently and dramatically, we still are going to have to deal with a deficiency of acetylcholine and the symptoms it produces. So the deficiency of acetylcholine is not the cause of Alzheimer's disease.

This is really step 2 in the research on Alzheimer's disease. The deficiency of acetylcholine results because the cells that should be producing it are dying, degenerating at a very slow but progressive fashion, and it is the death of these cells that results in the chemical deficiency. We now have to move on into step 2 of the research to find out what is the cause of this cell death.

I think progress in this area is certain to come through the application of molecular biological techniques, and Charles has been among the leaders in this field, in showing that you can take some of the most sophisticated recombinant DNA technologies and apply them to the postmortem human brain.

There is a specific area, I think, in which Alzheimer's disease research has been really deficient, and that is in studying the molecular genetics of the disease. The genetics of Alzheimer's disease in general is far from simple. It is a very complicated pattern of familial disease sometimes, and apparently isolated cases here and there most of the time. Nonetheless, there are families known in this country and overseas in which the disease is inherited in a very clear and very simple pattern. A national effort to find these families and to enlist them into the research is essential.

This has been done in research on Huntington's disease with enormous success. Huntington's disease and other of the degenerative neurologic diseases are much rarer than Alzheimer's disease. Nonetheless, for the last 4 years the extraordinary energy of people like Nancy Wexler and James Gusella have been devoted to finding, collecting, analyzing samples from Huntington's families, and late last year their results bore fruit. They now know with a considerable degree of precision where the gene causing Huntington's disease is.

Such an effort in the field of Alzheimer's disease has barely begun. There is no scientific barrier preventing us from precisely reproducing the success of the Huntington's project——

Mr. GORE. How should we proceed on that?

Dr. DAVIES. My feeling is that the most important person in the Huntington's disease effort is the coordinator, the person who finds the families and arranges for the families to participate in research. This can be terribly arduous work, especially in today's mobile society where members of one family might live anywhere from Hawaii to Honduras. But somebody has to trace these family members, find them, talk to them. We need, I think, a national coordinator. Gusella from the Huntington's project and I spoke about this matter, and the difference between Alzheimer's and Huntington's disease is one individual, Nancy Wexler, who coordinated the Huntington's effort with extraordinary energy and diligence. We do not have Nancy Wexler in the Alzheimer's project, but we sure could use somebody like her.

Mr. GORE. We will pursue that in our questions. Go ahead.

Dr. DAVIES. The other point about these studies is, as the Huntington's project has demonstrated, access to the cells that are derived from these patient samples to all investigators is vital. There are always issues of territoriality in any kind of research endeavor, but with the proper coordination, centralized cell storage facilities can make these cells available to anybody who has a bright idea and wants to try it out. This is very important. It is very important to avoid one individual limiting access to these kinds of resources.

The possibility of successfully applying the molecular genetic approach is strengthened by some recent work which essentially rounds out a link between Alzheimer's disease and Down's syndrome. This is a very hot clue to where the cause of Alzheimer's disease might lie. The link now works two ways. Individuals with Down's syndrome, who have an extra copy of the normal Chromosome 21, appear to develop Alzheimer's disease with high frequency if they live long enough. Over the age of 45 or 50, this frequency is anything from 50 to 90 percent, suggesting that something on Chromosome 21 means that development of Alzheimer's disease is almost inevitable. But there is a second link, when Heyman confirmed Heston's earlier work. That is, if you look at families with Alzheimer's disease, they are approximately three times more likely than normal to have given birth to a child with Down's syndrome, which suggests that these families have some kind of fundamental abnormality on Chromosome 21 that leads, on the one hand, to the development of Alzheimer's disease and, on the other hand, to an increased frequency of the Down's syndrome individual. So obviously, the place to begin looking for a cause of Alzheimer's disease is Chromosome 21.

This is an interesting issue, when you bring up molecular biology and Down's syndrome. It is true, as Charles said, that neurosciences tend to borrow heavily from other techniques, and I would like just briefly to quote a sentence from Julius Axelrod, the Nobel laureate in the neurosciences, which is from the current issue of Science magazine:

"Neuroscientists have borrowed heavily from molecular biology, biochemistry, biophysics, immunology, genetics, and cell biology to do their work." We have. We take recombinant DNA technology, which was developed largely in cancer research, funded by the National Cancer Institute. We now take monoclonal antibodies from immunology and use them as tailor-made reagents. It is interesting, too, in the Down's syndrome research, it has largely been supported by Child Health.

While I applaud the congressional attention to Alzheimer's disease, I think there are two points to be careful about. One is not to do too much directed funding. While it is nice to be in the favored position of working on Alzheimer's disease because that is where the directed funding is going, I do not know where the next technology is going to come from, whether it is going to be from Child Health and Down's syndrome or some obscure allergy lab somewhere. I do not know. I think that the national coordination of Alzheimer's disease research would be very useful, but I think if additional funds are available, they should go to the peer-reviewed investigator-initiated grants in all basic laboratory research, all basic medical research. It is in the laboratory that Alzheimer's disease will be conquered, not in the clinic or the home, laudable as the efforts are in those areas. It is in the laboratory that we will solve this problem, but I do not know which laboratory.

Thank you.

[The prepared statement of Dr. Davies follows:]

STATEMENT FOR U.S. HOUSE OF REPRESENTATIVES, COMMITTEE ON SCIENCE AND TECHNOLOGY.

September 20th, 1984.

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Over the last seven years, considerable progress has been made in understanding the neurochemistry of Alzheimer's Disease. It is now clear that this condition is a real and rather specific disease, and certainly not a generalized destruction of brain tissue. Numerous reports have confirmed that one of the major targets of the disease process is a group of nerve cells that produce the chemical acetyl choline. It is also well established that the production and release of acetyl choline in brain is extremely important for functions such as memory for recent events, learning new skills and orientation. Our own recent studies appear to confirm that normal levels of intellectual function are impossible in the face of major deficits in the ability to produce acetyl choline. Thus our current view that at least some of the major symptoms of Alzheimer's Disease are the direct result of a deficiency of acetyl choline. In some patients with Alzheimer's Disease, other chemical deficiencies may be found, particularly in the late stages of the illness. These include reduced levels of somatostatin, substance P and noradrenalin. However, these deficits are neither as severe nor as consistently found as that of acetyl choline, and current research does not provide clear evidence that these deficiencies play a role in causing the major symptoms of the disease.

The above paragraph raises two important points:
1. If the symptoms of Alzheimer's Disease are the result of an acetyl choline deficiency, then some treatment of the symptoms should be possible. Unfortunately, this is not a simple matter, because of the complex nature of nerve cells that produce acetyl choline. However, the first very tentative steps in this direction may have been taken: for the first time, a drug which enhances the action of acetyl choline has produced reproducible and statistically significant improvements in Alzheimer patients. It must be stressed that the benefits of currently available drugs are statistically, but not clinically significant. The patients do better, but do not improve enough to help their families or themselves in any real sense. Further research on the development of more effective means to treat a deficiency of acetyl choline are clearly warranted.

2. Even if more effective drugs were available, we would still have less than half the answer to Alzheimer's Disease. This is because the deficiency of acetyl choline is the result of some attack on the nerve cells that produce this chemical: this attack is unlikely to stop just because we find a good substitute for acetyl choline. To conquer Alzheimer's Disease, we must find the cause of the degeneration of acetyl choline-producing cells, and either prevent or delay its action. Neurochemical research has identified a major target of Alzheimer's Disease within the brain; we must now move forward to find out what is hitting that target.

It seems likely that major developments in this field will come as the result of the application of the techniques of molecular genetics to the study of Alzheimer's Disease. While the genetics of Alzheimer's Disease in general are far from simple, there are families in which the disease is inherited in a clear and simple pattern. A national or perhaps international effort should be mounted to find, document and enlist members of such families into research on this disease. Blood cells from these families should be available to all qualified investigators, to facilitate sharing of research resources and results. The appointment of a single dynamic individual to coordinate such a program would be of great help. An effort almost identical to that proposed above has been mounted for research on Huntington's Disease, and this has been functioning for some years. Late last year, the efforts of Drs. Nancy Wexler, James Gusella and colleagues resulted in a spectacular success: the description of the location of the gene causing Huntington's Disease. There is no scientific barrier preventing the application of precisely the same techniques to studies of Alzheimer's Disease, and no reason why they should not be equally successful. The immense and growing power of recombinant DNA technology promises to revolutionize research on Alzheimer's Disease and other neurologic disorders.

The possibility of successfully applying molecular genetic approaches to Alzheimer's Disease is supported by recent work from Heston in Minneapolis and Heyman at Duke. It has long been evident that individuals with Down's Syndrome (also called Mongolism) appear to have a very high risk of developing Alzheimer's Disease if they live past the age of 40 years. Although the clinical symptoms of Alzheimer's Disease are not always readily apparent in an individual who is severely mentally retarded, the chemical and pathologic changes seen in middle aged victims of Down's Syndrome are clearly those of Alzheimer's Disease. We know the cause of Down's Syndrome: it is the presence of an extra copy of chromosome 21. This appears to be enough to result in the almost inevitable development of Alzheimer's Disease, if the individual lives long enough. But the link also works the other way around: Heston and Heyman have shown that a woman with Alzheimer's Disease is about three times more likely than normal to have given birth to a Down's Syndrome child. This suggests that a fundamental cause of Alzheimer's Disease may lie in an abnormality on chromosome 21, even where inheritance patterns are not clear.

If this committee would like specific recommendations (and even if it would not), my position is as follows:
More support for basic research is essential. Alzheimer's Disease will be beaten by laboratory research. Government should be careful to avoid too much directed research support, because it is impossible to predict where

the next key technique or development will come from. Recombinant DNA techniques were largely born in cancer research, and monoclonal antibodies from immunology, yet both serve research on neurologic disease as though custom made. Child Health has supported most of the research on Down's Syndrome, which is now so relevant to finding the cause of Alzheimer's Disease. It is extremely important that additional funds, if available, be directed to supporting peer-reviewed, investigator initiated grants in all fields of basic medical research. This, with some national coordination of efforts directed specifically at major public health problems such as Alzheimer's Disease, would ensure success in our endeavors.

Mr. GORE. Thank you very much.

Let me start off, if I can, by asking you to help me understand in layman's terms, if that is possible, exactly what point you have arrived at now in your understanding of it. When you talk about neurotransmitters, what you are talking about are chemicals that carry messages from one cell to another cell; is that right?

Dr. DAVIES. That is precisely correct.

Mr. GORE. Those neurotransmitters are proteins?

Dr. DAVIES. No, they are in general much smaller molecules.

Mr. GORE. Here is as far as I have gotten. You help me get further than that. You discovered, Dr. Marotta, using the technique of studying frozen brains, which you found actually were useful in that they retained some of the functions of RNA and DNA that people thought maybe they did not before, you found a particular deficit, a particular thing wrong with Alzheimer's patients?

Dr. MAROTTA. I see you are trying to fit this into a picture.

Mr. GORE. I am trying to draw a simple picture; that is right. Now let me take it a little step further. DNA is, of course, the basic blueprint of life. RNA is produced by DNA, and RNA is essentially a "soft copy" of DNA; correct? For those who use personal computers, it would be like making a copy of the memory and using that when you keep the hard memory intact, like a Xerox copy?

Dr. MAROTTA. That is true.

Mr. GORE. The DNA produces the RNA, which has the same coded information but it is more perishable; is it fair to say that?

Dr. MAROTTA. Yes.

Mr. GORE. The RNA then creates proteins. It tells the cells how to function normally; correct?

Dr. MAROTTA. Correct.

Mr. GORE. You found that the RNA of Alzheimer's patients was greatly reduced in quantity because it was apparently being consumed by an enzyme; right? Now, the reason it was being consumed by an enzyme is that another enzyme that was supposed to eat up the first enzyme was missing.

Dr. MAROTTA. Generally, that is true.

Mr. GORE. So enzyme No. 2, which was supposed to eat up enzyme No. 1, was not there to do its job. So enzyme No. 1 is just running wild, like Pac Man, eating up the RNA. Therefore, the message was not getting through to the cells, and the cells were not operating normally?

Dr. MAROTTA. Generally, that is true.

Mr. GORE. How does that fit in? Where in that simplistic description is acetylcholine?

Dr. DAVIES. I wish I knew the answer to that one. We are still working at somewhat different levels, Charles and I, in this problem. We cannot fit our observations together specifically into a simple picture. We do not know the significance of the decreased amount of RNA or decreased activity of the RNA for the production of acetylcholine.

Mr. GORE. Wait a minute. How do you get the neurotransmitters from the RNA?

Dr. DAVIES. The enzyme that produces acetylcholine is made from RNA. The RNA directs the production of the enzyme that produces acetylcholine.

Mr. GORE. If the RNA is reduced in quantity, then presumably one possibility is that means it is not producing enough of the enzymes that have to produce the acetylcholine?

Dr. DAVIES. Yes.

Dr. MAROTTA. That would be one possibility.

Mr. GORE. That is only one possibility. What are the others? Are there other leading possibilities?

Dr. MAROTTA. What we have observed may be a way of looking at other, more fundamental processes which lie at the DNA level. I think it is unlikely that we have found the key process in Alzheimer's disease; I do not believe that. I think that if we can find this process, there are many other processes that are generally grouped as regulatory control mechanisms, which function to establish the normal levels of the enzymes that are involved here and all proteins.

Essentially, we do not know the exact relationship between our findings and this particular enzyme, although we have testable hypotheses, and you have given us a very reasonable one. We do not know what goes beyond that, back to the DNA level, although I think it is unlikely that DNA is not involved. Ultimately, we do not know the trigger for all these to occur in the first place.

It could be a sequence of events that occurs by as yet an unknown process, and Peter is measuring one aspect, and I am measuring another aspect of it. Typically, in biology, it is not usually that simple a cause and effect because of the complexity of cellular life. There are always other control mechanisms which function to regulate the very many macromolecules in the cells. I believe we have just really the tip of the iceberg that is being observed here. So we have the hypothesis that you are suggesting, but I also believe—and I think it is hard to disagree with it—there are also many other possibilities that are much more complex than the ones that we have investigated so far.

Mr. GORE. I interrupted you earlier in my effort to try to match that simplistic description with the line of research you have been taking, but go ahead with what you were going to say.

Dr. DAVIES. I think Charles and I are really working on the same problem from different angles. In fact, we can put it this way. When you look at the brain in particular, you are looking at such a hideously complex organism or piece of an organism that you really do not know where to begin with a diseased entity. What you have to get is some handle on what is happening in the brain. My own focus was simply to find out which cells were always affected by the disease, because that is where we can start looking for the cause of a disease. If this group of cells is only affected in 25 percent of the patients, that, to me, is not a fundamental issue in the disease; it is another problem that I will deal with when we have gotten rid of the first one. I want something that is affected in every patient I look at, so I can know where to look for the target. I have the target, and I am looking for what hits the target.

I think the press attention to Charles' work was a little misplaced. I think Charles would state this, but not in the same way that I would. The finding that the RNA is less active and is being eaten up is a very interesting one; we do not understand its significance. What Charles did some years ago was to sit down and say, "Can we do molecular biology on brains from Alzheimer's patients, and that means on autopsy tissue." I thought about the same thing about 7 years ago, too, but I was discouraged by my colleagues from ever trying it because they said if you ever put that in a grant application, you are dead. It is ridiculous. It is a silly experiment. I think Charles still suffers some resistance from the classical molecular biologists who say, "You cannot do that on this." You can, and Charles has shown that you can. That is far more important than the observation on chewing up RNA, in fact, because it shows we can do it.

Mr. GORE. The research methodology is, in itself, a significant breakthrough?

Dr. MAROTTA. Yes, I would agree with that. This touches on some other comments that were made before. There is some resistance on the part of granting agencies and colleagues to trying new and ridiculous ideas. I will give you an example of this. When I decided to do these experiments, I was told by colleagues that they could not be done because everyone knew they could not be done. Of course, that was not true because there was nothing written on it.

A comment that was made to me at that time is one for which I still have no answer. It is: If such experiments could be done, then someone emminent would have done them already.

Dr. DAVIES. Now someone has.

Mr. GORE. That is right.

Dr. MAROTTA. It points out, there is resistance to trying new ideas. This bears upon a comment Peter made which I think is very important. Although I would like to see a directed focus toward Alzheimer's disease research, granting agencies should not prescribe the methodology to be used. I think funds should be available and that you should leave scientists alone to do whatever they want to do. The cancer funding has opened up new worlds of basic research because the money was there and people just followed their own instincts. Cancer research has revolutionized biology, not just cancer research. You simply cannot predict where new ideas that work are coming from.

A very good example of this is that there are a group of enzymes that are the cornerstone of cloning techniques called restriction enzymes. These were discovered initially—although it is often forgotten—many years ago by Matthew Messelson, who was studying how viruses infect bacteria. It seems like a very big jump to go from E. coli to the major recombinant DNA laboratories, but it was a very simple observation he made. It was done on the basis of general funding, because he was interested in knowledge.

Mr. **Gore**. We want to explore the implications of your experiences for how we fund basic research and how the agencies keep out of your way to the appropriate degree. Let me ask just one more basic question, and then I want to recognize my colleagues. I appreciate their indulgence.

One of the significant things about your discovery, Dr. Davies, and correct me if I am wrong, is that you identified a relatively small area of the brain where the damage associated with Alzheimer's disease is located or focused. Before that time, the possibility was open that it was a sort of general, systemic order throughout the brain. Am I on track there?

Dr. **Davies**. But I must spell out the specifics. Otherwise, my colleagues in other areas will say I am claiming credit for things I did not do. I was one of the three original groups who reported the evidence of a deficiency of acetyl choline. When I first came across the observation, I did not know what these cells were. In fact, I did not know that for the next 4 years. It was largely the work of the Hopkins group, Coyle and Price, who discovered where these cells actually are in the brain.

Mr. **Gore**. Dr. Price was here yesterday.

Dr. **Davies**. I see. Good. They were represented.

The important thing about the anatomy and the neurochemistry coming together was not so much that damage is localized to the acetyl choline producing cells but that the damage is always there. There may be in some patients other areas of damage. That is true, and I am the first to admit that. But it is awfully hard to find really good consistent damage in every patient anywhere but in these acetylcholine producing cells. We have looked at hundreds of brains, and every single one has this damage. In fact, I tried to total up the number of brains which have been studied by 12 different groups around the world and in 30-odd publications, and it runs into several thousand I would guess by now. Everyone has this acetyl choline deficit.

Mr. **Gore**. So in summary, both of you would agree that the scientific answer probably lies at a level of detail to which we have not yet penetrated in our understanding of basic DNA and RNA functioning, and that more basic research in the microbiology and chemical functioning of these basic building-block elements is what is going to be most helpful in finding a cure?

Dr. **Davies**. Absolutely.

Dr. **Marotta**. That is correct.

Mr. **Gore**. Mr. Skeen.

Mr. **Skeen**. Thank you very much, Mr. Chairman. I would like to defer, if you would care to go ahead.

Mr. **Winn**. No, go ahead.

Mr. **Skeen**. This has all been a very interesting discussion this morning. We are going to grant Congressman Gore his Ph.D. in biology because we have gone through oncogenes, and now we are into this discussion, and he does an excellent job. I may spoof him a little bit, but I do have a great deal of admiration for his ability to grasp and then try to turn around and make this thing a very simple problem.

I would like to get back to what you perceive to be the problem with granting agencies in dealing with these grants, because both of you have alluded to this common problem. In our determination of what the disease is in grants, if there is one, we make a lot of mistakes in this body up here trying to be helpful. I would like to turn the thing around and say, "You tell us, if you were in our position, how you would handle the structuring of grants so that it would be open, so that you had the flexibility." From one extreme it would be to say, "Here is the funding. Do what you think best."

I am a little disturbed that within the structure and the ego-climbing of research that you still have scientists who say that it cannot be done or we've done that or you can't do that. It would be my view that you can do it, but how do we help you "can do"?

Dr. **Marotta**. Let me clarify by saying that neither Peter nor I are saying that the NIH has not done a good job.

Mr. **Skeen**. I do not mean to imply that. I am talking about the entire research structure. If we are going to handle grants, how best could we do it in your view?

Dr. **Marotta**. I would not change the peer review system. I believe that is effective.

Mr. **Skeen**. Keep it intact?

Dr. **Marotta**. Yes. I think it comes down basically to there not having been enough money available for the kinds of research that I wanted to do at the time that I wanted to do it.

Mr. **Skeen**. That is a common problem.

Dr. **Marotta**. It is a real problem. It sounds simple, but that really is the truth. In order to supplement my Federal grants, I have had to apply to private foundations, which rarely have the resources that are necessary. I think I would like to see more daring approaches to basic research.

Mr. **Skeen**. What do you mean by "daring approaches"?

Dr. **Marotta**. One scientist I know had proposed doing recombinant DNA experiments with postmortem materials almost 2 years ago. In his reviews, he was not not-funded; it was just left pending. He was told that these experiments are premature. Today, we believe that these experiments are necessary.

Mr. **Skeen**. You have to educate me a little bit, doctor, on the kind of reviews that you have to go through to obtain grants. It is not a common knowledge to most of us. I can gather from the conversation we have had this morning. So tell me, was this a peer review system that reviewed the research and gave this determination on this particular project?

Dr. **Marotta**. Yes. This is a general problem. I am using it as an example. You actually get a score on these grants.

Mr. **Skeen**. You get a report card?

Dr. **Marotta**. Right. It was still within the ballpark of potential funding, and scientists felt encouraged and waited. In the end, it came down to how much of the funds were available after the better scores were funded to then go on to the not-so-good scores, which sometimes included some of the more daring experiments.

Dr. Davies. Having sat for a year on one of the peer review groups for the Neurology Institute, I learned very fast the grant game.

Mr. Skeen. It is a game, too, I understand.

Dr. Davies. Yes, of very restricted availability of funds. On study section, you sit and review 110 grants in 3½ days and give each one a score. You know that there is a very limited amount of money available, and although that is not supposed to influence you, it does. So what you tend to fund, with a group of 18 people of varying persuasions around the table, are safe projects, projects that you know will produce, because you do not want to fund duds when there are good people out there who are not going to get funded because something that may be a dud or may not be getting funded. You know there are good, productive laboratories who are onto a sure thing, with good productivity, and that changes your attitude toward funding the more innovative and high-risk projects, what are described in study sections as "fishing expeditions."

Mr. Skeen. Let me ask you, then. It changes from a process of doing real research and what a researcher's opinion and his ability and a plausible approach then to one of what is more publicly accepted or one that has more PR pizzaz or something like that.

Dr. Davies. Let me be honest and tell you about my own case.

Mr. Skeen. I would like that.

Dr. Davies. I have been involved for the last 9 years in research on the acetyl choline system. I have, I think, a very good reputation in that system and can get quite readily funded, submitting grants to measure more acetyl choline in more different dementias. I do not want to do that. I want to become a molecular biologist and use the techniques to find the cause of Alzheimer's disease through the cells that produce acetyl choline. If I put a grant in to do what I am actually doing in the lab now, it would not get funded because the study section would say, "He has no experience, no training, and no reputation in the area of molecular genetics." I think that is entirely accurate.

Dr. Marotta. One of my recommendations before was to attempt to lure into molecular biology research people who already have a commitment to Alzheimer's disease, and Dr. Davies is a very good example of that. He is just having some difficulty entering that area.

I would like to give you an example of high risk experimentation that paid off. Several years ago, it was somewhat unlikely that Jim Gusella at Harvard would be able to find a genetic marker for Huntington's disease. It was through the efforts of Nancy Wexler, who spent a lot of time explaining this, both to the scientific and bureaucratic communities, that such things were possible based upon the information she was given from Gusella and his associates. It was high risk, but it was high risk based upon years and years of very good experimentation that was carried out by Gusella and his associates at MIT. It was high risk in concept, it was not high risk in technology. That is a very important distinction there.

It was a matter of selling the good technology that allowed that material to be funded.

Dr. Davies. We both, I think, are committed to the peer review system. The two things I think we would like to change in it, apart from the amount of money available, are the time period on grants because three years is ridiculous. We have currently in the lab five grants, all on fairly short cycles, and you do spend maybe 6 to 8 weeks a year writing grants and then another 9 months waiting to find out whether it is going to be funded, making calls to Washington, sending in supplemental information. It is an extraordinary waste.

Mr. SHEEN. I am astounded by Dr. Marotta's statement in his testimony here that 30 percent to 40 percent of his time—and I am assuming a higher percentage of your time——

Dr. DAVIES. It is about one-sixth of the time.

Mr. SKEEN. Preparing for grants?

Dr. DAVIES. Yes. In these tight days, you have to put the best grant together that you can. So you spend more time on it than perhaps you should.

Second, for the period of grants, I think there ought to be some pool of money which is specifically for high risk and innovative projects. Again, this cannot be a 1-year pilot grant. With 1 year, 3 months is gone and you have to submit the renewal. You get nothing done in that kind of period. It does not have to be a big pool, and it can be targeted, but it is a pool that currently does not exist. You probably need about $10 million a year for such a pool.

Dr. MAROTTA. High risk does not necessarily mean low quality. In the peer review system, the scientists who sit on the panels know if an applicant is a solid scientist. If he has a high-risk project, that should be taken into account in view of his past history. So he would not be wasting money on foolish enterprises, because the scientist would be reviewed as much as the project.

Mr. SKEEN. I thank you both very much, because that is the kind of insight that we need. If we want to help you, which is why we are here and the result of these hearings, we hope, produces the kind of help from the legislative body of this kind, we would like those kinds of recommendations, and we would like your candid approach to this thing. That gives us an insight as to what happens to the problem once we leave it. We can draw up a piece of legislation here, and give you something to work with. We can also make the appropriations, but we cannot go through the system unless we know it is working well. If the peer review system is working, fine, except that it needs to be loosened up and we need to expand the time—those are concrete suggestions that we can do something with here. I thank you both very much, and thank you, Mr. Chairman.

Mr. GORE. Mr. Winn.

Mr. WINN. Thank you, Mr. Chairman.

First, I would like to bring some good news to the committee. I serve on the Office of Technology Assessment board, and a little earlier this morning we approved a new project called an assessment proposal named "Disorders Causing Dementia," which would include Alzheimer's.

Mr. GORE. Would my colleague yield?

Mr. WINN. Certainly.

Mr. GORE. I am delighted at that news. Our subcommittee and full committee formally requested that on the first day of these hearings. I was very comforted when we made this request, knowing that you served on the OTA board. I am glad that approval did come.

Mr. WINN. Thank you. I thought the committee would like to know that.

Let me change the subject a little bit. Again, all of us appear to need education on the subject a little bit. Much has been reported in the media about new drugs which offer the prospect of treating some of the major symptoms of Alzheimer's, for example Physostigmine and Naloxone.

Dr. DAVIES. Physostigmine is the drug I referred to in my written report, which is an experimental drug which has as its action to slow down the breakdown of acetylcholine in brain and, incidentally, in the rest of the body, too. It has been used, to my knowledge, in two very good clinical trials on Alzheimer's patients. They were very good in that these trials were blind, the doctor does not know what drug the patient is getting, whether he is getting the drug or the placebo, and both trials were conducted by very good people. The results do show improvement in Alzheimer's patients, improvements in tests of memory—how many words do you remember—improvements in inappropriate responses to the testing.

These results are really not significant to the patient or the family. They do not see the patient say, "I am better. I am going back to work tomorrow." They do not really see any major improvement in the patient at all. In fact, most of the families cannot tell if the patient is improved. This is just the first step. This drug is not the ideal drug for treating Alzheimer's disease, and it is actually quite a dangerous drug if used without due care. But the significance of this is scientific rather than clinical: we can make these patients improve if we use drugs that facilitate the action of acetyl choline. That is a very good start.

Mr. WINN. How long have you been using that Nalaxone?

Dr. DAVIES. I have not personally been using it.

Mr. WINN. How long has it been available?

Dr. DAVIES. Approximately 2 years in experimental studies. It is not the answer to Alzheimer's disease.

Mr. WINN. What about the other one?

Dr. DAVIES. Naloxone, I believe, is now in proper trials, double-blind trials. It has not been used extensively in well-controlled trials as yet. These trials are on-going. I have not heard very encouraging results with Naloxone, but I do not know the results of the trials. It is too early to tell.

Mr. WINN. Dr. Marotta, before asking questions regarding your particular study, I would like to ask questions about autopsies in general. We touched on that, and it has been mentioned. We know that autopsies in this country can be performed only with the consent of the family or the next of kin. Do you believe that the autopsy rate among the elderly is the same as that of lower age groups? In other words, are we short elderly brains?

Dr. MAROTTA. I do not have any figures to give you, and I am not certain of the answer to that. I do know that because the public is becoming more aware of Alzheimer's disease as one of our great health problems, more and more individuals are now donating their brains to brain-collection centers. This has been the experience at McLean Hospital and it is probably true at Einstein as well.

There seems to be not enough donation of nondemented brains, of normal brains which we can use to control the experiments with the Alzheimer's disease brains.

Mr. **Winn**. That is a very interesting statement.

Dr. **Marotta**. It just does not seem all that important to families with healthy elderly to donate that material.

Mr. **Winn**. I think that is a very important and a very interesting statement. I can probably understand that the next of kin of someone with a normal brain is not emotionally desirous of having an autopsy. Possibly there needs to be some educational work done from the research field and from the medical field.

Dr. **Davies**. It is really an acute problem. We do function as one of the brain-collection centers, and we have probably 120 brains from Alzheimer's victims and probably less than 10 normal brains, which greatly hampers research. You cannot conduct experiments on what is abnormal unless you know what is normal, and that is a very major problem.

Mr. **Winn**. I appreciate your bringing that up. The reason that I was asking that question is, some of the people have testified yesterday said that up to 20 percent of the cases diagnosed during life as Alzheimer's disease turned out to be not so in the autopsies.

Dr. **Davies**. That is one of the very important functions of the brain-collection centers, to provide in addition to tissue research a pathological diagnosis of what in fact was wrong. It is true, there is a significant misdiagnosis of Alzheimer's disease, even in patients who are seen at the very best centers. This becomes important particularly when there is a suggestion of inherited Alzheimer's disease. Families run very scared without, in many cases, ever having had an autopsy on a member of the family. So they really do not know what it is that is apparently being inherited.

We have a case recently of a family from Florida who sent the first member of that family to die with this crazy disease to autopsy. The brain came to us, and the lady does not have Alzheimer's disease. She had a series of strokes.

Mr. **Winn**. Did she have some form of dementia?

Dr. **Davies**. She had several small strokes. In fact, it was the cause of her dementia. But this illustrates that the family was living in fear of inherited Alzheimer's disease, and in fact there is no proof that there is Alzheimer's disease in the family. So that is another useful service.

Mr. **Winn**. How would either one of you or both of you recommend that we do a better job of educating the public as to the need for more autopsies, both with diseased brains and with normal brains. How should we proceed with that? How could we proceed with that?

Dr. **Marotta**. Of course, there is an impetus to families with an Alzheimer's victim to become involved. There is much less impetus to study the inheritance of health. However, a major aspect, in my view of Alzheimer's research is studying normal aging. In fact, that is the focus of my laboratory. I am not really certain how you would present this to the public and have them believe that it is very important. This is a new problem, and it really hasn't been addressed before.

Dr. Davies. One suggestion that we have been discussing is this. Normal individuals die on medical wards or surgical wards and not in the neurology service, where autopsy rates tend to be rather good and on medical wards they tend to be fairly miserable. One of the issues that might be handled through the National Institutes of Health is for the National Institutes of Health to urge that medical studies and surgical studies that it provides funding for should make every facility possible or available for autopsy on patients who die. This is not something that is done. In fact, some of the major longitudinal studies—the Framingham heart study, the Baltimore longitudinal study, for example—make no provision whatsoever for autopsies. I think this is an awful waste.

Dr. Marotta. We now, as part of our program in aging, have addressed this problem on a smaller scale. Perhaps our experience and our planning may be somewhat useful. There is in Boston a long-term longitudinal study of normal aging, carried out in one of the clinics in east Boston. That study is being conducted by Dr. Denis Evans, in collaboration with Dr. Marilyn Albert at the Massachusetts General Hospital. These individuals in the community are followed for many years and given neurologic and psychiatric evaluations as part of the normal aging study.

What Dr. Evans discovered was that a number of these patients were being brought to autopsy for other reasons in the local hospitals. This information somehow had not gotten transmitted back to the clinics. It is a very large population; thousands of individuals are involved. So my colleagues designed a study to track the patients, which amounts essentially to merely discovering who becomes admitted to our largest hospital, the Mass. General Hospital, and to see if these individuals were part of the east Boston study. So in fact, families are not asked to have the brains donated. We are merely discovering those which are normally brought to autopsy for other medical reasons, and we expect that this may actually solve our problem, in terms of normal controls.

Mr. Winn. That gives you a better supply.

Dr. Marotta. It gives us a better supply. It would not solve the overall problem. It would solve the problem for our laboratory.

Mr. Winn. There must be some type of an educational process that we can develop. It would have to be a cooperative effort, certainly, and we would certainly have to get the help of the medical journals and also the public press.

Dr. Marotta. The lay publications, yes.

Mr. Winn. One of you mentioned transplants. I would just like to point out that organ transplant legislation has been passed by the House and the Senate, but it is hung up in the conference right now. We are hopeful that if we can get it out of conference, that might create an increased awareness for the need of transplants. I think that has been pretty well publicized, because more and more, many families are confronted, directly or indirectly through friends or family, with transplant needs.

I would also like to point out, just for your education, that this morning on the technology assessment board, we also approved an assessment proposal for "Life-Sustaining Technologies and the Elderly," which certainly has been in the press for the last 4 or 5 years. Again, I am aware that these studies do not really make any final decisions, and that was not the intent, but possibly they could be helpful in the awareness problems of these two studies that are going to be made. We talked about peer review. I think you are aware that many of your colleagues sit in on some of these boards that help make some of these decisions. So we are gaining, and we are making some strides.

It is not your field, and it may be kind of a dirty trick, but do you have any recommendations as far as what type of clinics—do you all become involved in clinics as such, or do you go to any of the clinics to see Alzheimer's victims and what kind of treatment they are getting, or do you make any recommendations, or do you have any peer boards that sit in on any of those. I just do not know how they work at all.

Dr. MAROTTA. The medical community in Boston has banded together somewhat for Alzheimer's disease research. The people involved are bringing together the clinical facilities at the Massachusetts General and the Beth Israel Hospital to try to pool their information, in terms of the diagnosis and treatment of Alzheimer's patients. MIT and McLean Hospital are involved in this. The clinicians are establishing standardized procedures for reviewing patients and treatment, and the individuals who are in these clinics eventually will be introduced to the idea of brain donations through their families at some point. So efforts are being made to pool information in this geographic area, to pool patients, and then eventually to make these brains available to investigators. One of the stimuli for this was the creation of Alzheimer's disease research centers, which is a program of the National Institutes of Health, which was a great impetus to have these individuals come together. We are very hopeful that we will achieve that funding and have more uniform methods of collecting brains based upon the similar ways patients are treated in clinics rather than different methodologies.

Mr. WINN. Do you share brains from lab to lab? Do you send them around once? The family is given the right for an autopsy—let's say it is at Boston—and they do their work and they make their report to the family. I know how that works. But do they then send it on to some other labs for additional research?

Dr. MAROTTA. Under the best and most altruistic of circumstances that in fact does occur. When brain banks receive Government funding, they are required to make brain tissues available to all interested investigators. McLean Hospital does have the oldest brain bank in the East for studying schizophrenic and Huntington's diseased brains. In fact, those tissues are made available on a national basis to any established investigator. We are hoping now that the same will be done with Alzheimer's brains, through what is referred to as the Alzheimer's disease research center, which will be located primarily at the Massachusetts General Hospital in Boston. Those brains, if that center is funded, will be made available to all investigators in the Greater Boston Area.

Mr. WINN. Is the brain and the tissues not only available for additional research but is the report, the original report that was sent to the family or the next of kin, passed on, too, to show what the first investigation found?

Dr. MAROTTA. I believe it is. A part of the center is to have the families and the communities involved in being supports for the center. Although I do not know the details of it, I know that has been addressed.

Mr. WINN. Do you care to address that in any way, doctor?

Dr. DAVIES. We have certainly sent tissue to as many people as make reasonable requests for it and sometimes unreasonable requests for large amounts of tissues. Yes, there has been no denial of tissue that I know of to any investigator interested.

Mr. WINN. It has just been pointed out to me by staff that $3.5 million has been authorized for the Alzheimer's disease research centers in 1984 in the NIH bill. I believe before, as I was coming in, you discussed that, Dr. Marotta.

Of course, money and funding is a problem that we have mentioned here several times. It is obviously one of our big problems. But I want to commend you for the work that you have done. I think as far as this subcommittee and the full Committee on Science and Technology, which is only one of the many committees involved from Congress, that you have our support. We will do everything we can to help you keep up your good work.

Thank you, Mr. Chairman.

Mr. GORE. Let me pursue one question that my colleague asked you about, having to do with the availability of brains for study. Is it true that the autopsy rate among the elderly is far lower than among lower age groups?

Dr. DAVIES. I do not know the figures, but I would be very surprised if that was not true.

Mr. GORE. There was a letter to the editor in the New York Times a few days ago from, I believe, a researcher indicating that there was a real shortage of brains to be studied because of a very low autopsy rate. I am just wondering, you may not be involved with that aspect of it at all and may not know, but we will explore that with other witnesses. Specifically, we will explore whether or not there needs to be more public education and education within medical communities about the value of encouraging autopsies with Alzheimer's patients and with others so that the kind of research you all are doing can continue.

Because of your discovery, Dr. Marotta, about the value of this research methodology of using frozen brains, are there any implications you would draw about the need to freeze other brains of Alzheimer's victims?

Dr. MAROTTA. I am not sure I see quite what you are asking.

Mr. GORE. Maybe my assumptions are all wrong. Let me just take it step by step, very briefly. You found that it was extremely valuable to study frozen brains. Is there a shortage of such brains to study? Does that hamper other research similar to yours? If so, are there some recommendations that we ought to be making with respect to increasing the practice of freezing the brains of autopsied Alzheimer's patients?

Dr. MAROTTA. I believe you were out of the room when we discussed just a bit of this.

Mr. GORE. We had a vote over on the floor, and I apologize if I missed this.

Dr. MAROTTA. Let me just summarize what has been discussed, which I think addresses this. It is that as people become more aware of the magnitude of Alzheimer's disease as a problem and become less afraid of it in the family, families are allowing their afflicted individuals to have their brains donated for research. Now it is getting to the point at which we may be having a sufficient number of Alzheimer's brains to work with. The problem that we are encountering now is that we do not have enough normal brains to serve as controls in the experiments, nondemented non-Alzheimer's, from individuals who have a healthy old age. There is no impetus in the family to have these individuals autopsied, which is fairly understandable. It is just not brought to their attention. Public education would be very useful in this area.

Mr. GORE. Dr. Davies, you made two suggestions that I wanted to pursue very briefly. One is that your research indicates that although the cause of Alzheimer's is not at all linked to genetics, to inheritance, there may be an inherited predisposition to it, there may be an inherited cause. We just do not know. Nevertheless, the circumstantial evidence is a very powerful showing that there are some families that have a marked prevalence of Alzheimer's disease over generations; correct?

Dr. DAVIES. Correct.

Mr. GORE. And because of that fact, you believe it would be extremely valuable to the scientific research effort to have a study of such families or, rather, an effort to locate such families, as has been done already with Huntington's disease. I asked you earlier how we should go about it. What I was really getting at is, should we earmark a specific research line item to get somebody on board, tracking down those families, as was done with Huntington's?

Dr. DAVIES. I think that would be an excellent idea, personally. I do not know what the National Institute on Aging thinks.

Mr. GORE. Even though it is directed research?

Dr. DAVIES. Yes. That is the right kind of directed research.

Mr. GORE. OK. Maybe if the majority and minority staff can help us work up a proposal for the members' consideration, maybe that is something that we can come out of here with.

Second, in a little broader spectrum here, you said that it would be helpful to have better national coordination of Alzheimer's research. What do you mean there?

Dr. DAVIES. Again, taking the example from the Huntington's disease research, what was created was called the Huntington's Disease Center Without Walls, which was really a loose organization of the laboratories which received NIH funding and other funding for research on Huntington's disease, with the commitment to share research, resources, and results, not to wait to see your competitor's paper appear in Nature next week but to know what the results of the experiments were when the experiments were completed and submitted.

This is very helpful because it forces people to be a little less competitive and territorial in this kind of research, especially if you are going to talk about genetic research in Alzheimer's disease. There is probably a fairly limited number of families which are going to be useful for this kind of research—maybe 8 or 10 big families would be enough for the initial phase of the project. But if each one of those families belongs to a different investigator, is contacted by a different investigator, nobody is going to get anywhere.

Mr. GORE. The coordination you speak of, you were speaking of it with specific regard to the family research?

Dr. DAVIES. No, I think in more general terms, too. I see no reason not to follow the very successful model that Huntington's has shown us, that a center without walls where information and resources are shared by all those receiving funds from this common pool. We are all funded by essentially the same outfit.

Mr. GORE. How would we go about setting up that center without walls?

Dr. DAVIES. I think it would really be a fairly small coordination staff that could put this together, following the model that was very successfully used by the Neurology Institute of NIH for the Huntington's program. You need a small coordinate staff to identify those laboratories and outfits that are supported to work on Alzheimer's disease and to pull them together.

Mr. GORE. You are not talking about a bureaucracy. You are talking about a very small coordinating——

Dr. DAVIES. This would require probably four people, including a leading individual to go out and get the families.

Mr. GORE. Again, it may be possible that we could arrive at some bipartisan agreement on that. We can talk about it among ourselves. Certainly, if it was as successful as you indicated with Huntington's, this is something that we will definitely explore as a specific recommendation. If staff will make note of that, we will pursue that.

Dr. MAROTTA. A useful person to have input from would be Nancy Wexler at NIH.

Mr. GORE. Congresswoman Schneider has come in, and we will be glad to recognize her.

Mrs. SCHNEIDER. I regret that I had other committee demands on my time and that I missed your testimony. At this time, I have no questions, but will review your testimony at a later time. Thank you, Mr. Chairman.

Mr. GORE. We could spend a lot of time with this panel, because your testimony is fascinating. We could go on, but we have many other witnesses. We are just really honored that you would come and spend time here. You are representative of a whole group of scientists who are devoting their lives and careers to work that means more than words can describe to many millions of Americans. We are all very grateful to you. You are doing terrific work. Thank you for being here.

Before going to our second panel, I want to recognize Congressman Bill Lowery, a member of the committee. We put your statement in the record already. If you would care to summarize any portion of it, Bill, feel free. We are delighted to have you and enjoy working with you, and we will recognize you at this time.

STATEMENT OF HON. BILL LOWERY, A U.S. REPRESENTATIVE FROM THE STATE OF CALIFORNIA

Mr. LOWERY. Thank you, Mr. Chairman. Again, my apologies that my commitments did not allow me to get out of them early enough so that I could be here at 9:30. You do have my testimony in the record, so let me just summarize a couple of things on this one member's interest in Alzheimer's disease.

I have considered myself a reasonably sophisticated member of the community, but I did not know a thing about Alzheimer's disease until 3 years ago. A lady by the name of Joy Glenner walked into my San Diego office and shared with me its devastation, affecting over 2 million Americans, the fourth most common cause of death for those in our population over age 65. There are probably $30 billion a year in nursing home costs to the Nation. It is a devastation that I was unaware of and felt we needed to do far more to educate the American people. I think recognition is the first step toward any improvement or cure.

We have begun to do that, not only with HHS targeting it but with the Congress passing several resolutions and having weeks and now months—November is now Alzheimer's disease month—so that recognition can improve and continue, so that more public as well as private funding will flow so that we can find a cure and a treatment.

It strikes me that there are two major issues that we need to deal with. The first is money toward research for finding that cure and treatment and, second, alleviating the financial and emotional price tag that devastates families that have someone afflicted with Alzheimer's disease. More and more I think is beginning to be done. Not as much because I do not think there is ever enough resources to direct but far more than ever before. The fact that we have doubled public financing through Federal funding of research just over the last couple of years is encouraging.

Senator Heinz and I recently appeared on a nationwide program where a film was shown, "Whispering Hope," that I think is about the best thing that has ever been done on Alzheimer's. It was shown in some six cities. The scope of the questions that came in, not only to Senator Heinz and myself but other panelists, the experts in the medical care field, demonstrates that there is a sense of helplessness out there that we really need to address. Although I am confident more and more progress is being made on the research end, it is the current treatment that is quite troublesome, particularly since private health care programs, as well as public health care, do not recognize the so-called custodial types of treatments, be it in areas of alcoholism or Alzheimer's or Parkinson's disease.

We need to come to grips with lower cost treatment. Some of the innovations that have been suggested are having the area agency on aging pilot programs to capitalize on existing community-based services, or studying ways to maximize an older person's assets, perhaps an IRA established specifically for long-term health care, or tax incentives for nursing homes to accept more Alzheimer's patients, or tax credits for those who take care of victims in their homes in adult day care facilities, as opposed to the very expensive forms of traditional institutionalization of Alzheimer's patients.

There is so much that needs to be done. I think we are now recognizing that. The private sector is responding so well in my own home county of San Diego. The Alzheimer's disease and related disorders has a day care center which, in many ways, has been a model for other centers around the country, where for a nominal fee of $20—it started out at about $15—families of Alzheimer's disease patients can be assured that their loved ones will be cared for between the hours of 7:30 and 5:30 in a home-like setting that allows the family member to run errands, take care of business, or even just to relax and get a much needed rest.

I think the fact that there are now some 36 States, and by the end of 1984 there will be over 100 of these chapters in the country, is a very good beginning for self-help, of individuals banding together to help fight this devastation.

As we know, the costs are very great. The impacts upon the families are severe. I know that we have devastating concerns on the future of keeping public health care funds financially solvent. So to look at possibly added requirements of custodial care will be difficult. But more emphasis upon less costly forms of care, of home health care and other alternatives to institutionalization that are community-based home-oriented respite-type options I think clearly is an area that we need to focus on, not only because of the humaneness of the treatment but also because of the far lower cost.

I do not have any earth-shattering breakthrough solutions, other than to say that I compliment you, Mr. Chairman and this committee, for these hearings. I know we are all pitching and trying to find that cure and to have a better form of treatment. I am optimistic that we are going to win the war on this devastating disease.

Mr. GORE. Thank you very much for your statement, for your concern and activity on this. We will certainly look forward to continuing to work with you on this. Our committee has a special obligation, I think, as the Science and Technology Committee, to try to see that the Government does a better job of fulfilling its role. We will certainly look forward to continuing our work together.

Mr. LOWERY. Thank you, Mr. Chairman.

Mr. GORE. Thank you very much We appreciate it.

Let me call our second panel now: Dr. Leonard Heston, professor of psychiatry in Minneapolis, and Dr. Elias Manuelides, professor of pathology and neurology, at Yale, in New Haven. If the two of you would join us at the witness table, we would certainly appreciate it.

Without objection, your prepared statements will be included in the record in full. We are honored that you could join us today in our effort to try and deal with this problem. Dr. Heston, we will begin with you. We want to welcome you again, thank you for being here, and invite you to proceed.

STATEMENT OF LEONARD L. HESTON, M.D., PROFESSOR OF PSYCHIATRY, UNIVERSITY OF MINNESOTA, MINNEAPOLIS, MN

Dr. HESTON. Thank you very much. It is an honor.

I am a professor of psychiatry at the University of Minnesota, and for several years I have been engaged in research mainly involving families from a genetic point of view in Alzheimer's disease but also caring for many people with Alzheimer's disease in our clinics and hospitals.

I would like to start with a slide, if I may, to just briefly summarize some of our experience over the last several years.

I want to make a very simple point here about the nature of this disease. With most of the diseases I have illustrated there—with Huntington's disease and Pick's disease, which is another dementing disease, and rheumatoid arthritis, schizophrenia—many afflictions of the adult population in particular tend to cluster around the average age of onset. We have a bell-shaped curve, with the maximum age of onset as depicted on the lower ones.

On the other hand, Alzheimer's disease, which I have abbreviated there as dementia of the Alzheimer's type, seems to increase with increasing age. This is much unlike most diseases. It is more like occlusive vascular disease or the fallout of nephrons in the kidney or the loss of compliance in lung tissue. It is somehow related to aging.

No one knows enough about the frequency of these diseases to be able to fill out the areas under those curves and give an accurate representation of numbers. But for the time age 80 or so is achieved for dementia of the Alzheimer's type, we are looking at a frequency that is running some 20 to 30 percent of the surviving population.

The next slide, please. I want to make a simple point here. If you will look at the average survival and ignore the rest of it for the moment, this disease is much like many other diseases that afflict humans. It is called in genetic circles multifactorial, which in certain respects hides many things. A very complicated picture emerges. The point I want to make here is, like many diseases, the earlier the onset, the shorter the average survival. It starts out rather low in our experience with cases in Minnesota, and then the survival increases through the ages 50 to about 70. There is a pretty predictable about 8½ years of life remaining after onset. This suggests that the earlier the onset, the more severe the disease; severity in general is indicated by a decreasing period of survival.

In this, Alzheimer's disease takes its place with many other human afflictions. The average survival drops off later, because surely death from competing diseases—that is, Alzheimer's disease is no protection against heart disease or cancer or some other afflictions.

Next slide. This shows the same phenomenon, only in graphic form with statistical errors indicated by the vertical lines. This looks like a very lawful phenomenon, and in fact I think it is, with the survival slowly increasing and then decreasing again. Below it, the onset because of increased severity and then falling off again because of death due to competing causes.

The next slide. This is our actual experience with relatives of persons with proven Alzheimer's disease, cases proven by autopsy. You can see siblings, parents, second degree relatives, or one degree removed—that is, aunts and uncles, grandchildren and so on—and then estimates of the frequency of the disease for the general population. If you look down at the bottom line of the graph, the ages are indicated in 5-year intervals.

There are two or three points I want to make. No. 1 is, really to talk about the risk, one has to talk about an age-specific risk. It makes a great deal of practical difference whether one is talking about a risk of some 3 or 4 percent it looks like to me here at age 60 to 64 for a parent—3 or 4 percent at age 60 to 64, as compared with a much higher risk of 20 percent at 80, but there is a huge practical difference between 60 and 80. Therefore, the risks I think must be cited as age-specific.

The lines are pretty straight, which means there is an even increment for each age interval. If you look at the general population on the far right, it has roughly the same kind of shape, with a little bit of imagination. What it appears like from data such as those is that the risk is actually increasing, being shifted toward the left—that is, toward earlier ages; the people who are predisposed because of familial Alzheimer's disease are getting the disease at earlier ages but are getting it at about the same kind of rate as the general population, just younger.

Next slide. There are certain families which are at much greater risk. These are families—and this is also true in general in medical genetics—where the proband, who is the person through whom the family is located, can be presumed to have severe disease because the age of onset is low. In this case, I have divided it less than age 70: less than age 70, relatively severe disease.

Another indicator of severity is having an affected relative, one affected relative, and that in general will increase severity and multifactorial genetic diseases as it does here. If one looks then at the lowest line, which is the general population, those persons who have an older onset relative and no other person affected in the family, those two lines are hardly distinguishable from one another; the risk is not very much greater. The families that are at very much greater risk are represented in the next two lines, and those are cases where the person through whom the family is located has severe disease, early onset, and most severely affected at all, the proband or the affected relative.

In those cases, looking at the percentage risk in the vertical axis, 30 to 35 percent by age 75, 79. These are families that have something to worry about.

Next slide. This is a severely affected family. This one happens to be one I can show a lot of slides with no other family members affected at all. This happens to be a dramatic and severely affected one. I want to point that out. This family has two diseases: dementia of the Alzheimer's type, as I have indicated by the blue, and a series of cancers—chronic lymphocytic leukemia, CLL; multiple myeloma, MM; lymphosarcoma—and there are some families in which this kind of very dramatic clustering of diseases occurs. These are the families that I think are especially suited to recombinant DNA research and are especially targeted for biochemical and genetic research of all kinds.

Mr. GORE. Is this new information? We have not seen this before. We have heard about a possible link with Down's.

Dr. **Heston**. This is "in press" information. I have made this point before, but I have not shown these slides before.

Next slide, please. This is work we are doing with the National Institutes of Aging, with positron emitting tomography, as a way of assessing the progress of the disease, crude at the present. The point I want to make is, this is a living person who is known to have Alzheimer's disease because of his membership in a pedigree. He has a relative who has Alzheimer's disease known by autopsy; therefore, anyone else who becomes ill in these families is especially valuable for research of all kinds. This prevents a great deal of wasted effort, because you know the diagnosis as the study is being done.

The next slide. This is one person who returned to the National Institute of Aging from our study on three occasions, 3 months apart. If one looks from left to right, you are looking at the progression of the disease. In these studies, the reds and the yellows indicate higher metabolic activity within the brain; the more blue, the less. You can see, I think, proceeding from left to right across those slides, the metabolic activity becomes distinctly less.

Everyone knows that Alzheimer's disease is a progressive disease. This however helps us in a crude way, a beginning way, to set the rate. It is a linear decline. Knowing this particular individual well, I can say that this is not apparent from his clinical condition. When the first slide was taken there, the first two columns, he was working. He noticed his memory was failing, but there was very little other difference between line 1 and line 2. By the time you get to column 3, he had quit working. He obviously had failed quite considerably, but on the basis of the evidence from the positron emitting tomography, this is more of a lineal decline, a straight-line decline, as if there is a certain proportion of cells dying constantly, which is not reflected in what we see clinically.

I think those are all my slides. I have been, I hope, succinct. I will be happy to elaborate further.

[The prepared statement of Dr. Heston follows:]

SUMMARY

The dementing syndrome is associated with steadily progressive brain failure. Inability to recall recently learned material is usually its first sign. This is followed over the next years by increasing loss of both recent and remote memory, by developing deficits in abstraction and judgment, and by aphasia and apraxias; these manifestations of disease progress relentlessly until, after some 8-10 years of illness on average, a vegetative state is reached. Several discrete conditions are associated with the dementing syndrome; some of these are due to disorders extrinsic to brain, for example, endocrine or nutritional, and are known as secondary dementias. Excepting depression, which may rarely simulate dementia, the only common disease of brain tissue associated with the dementing syndrome is dementia of the Alzheimer type (DAT): DAT denotes a progressive dementia associated with the neuropathological changes first described by Alois Alzheimer in 1907. It will be the central topic of this chapter.

DAT

Until recently, DAT had been divided into two entities, Alzheimer's disease and senile dementia, by age at onset of illness; onset by age 65, Alzheimer's disease, after 65, senile dementia. Because the clinical phenomonology and neuropathology is the same in both entities, and there is insufficient evidence to warrant subdivision, DAT is now generally regarded as one disease consisting of what had been known as Alzheimer's disease, a rare illness, and senile dementia, a most common one.

Genetics and DAT

The evidence that genetic factors are important in DAT comes mainly from a few sudies based on relatively unselected probands supplemented by separate reports of about 50 families, no doubt biased because of their dramatic presentation and large size. A few pairs of twins have been described. While the work of defining DAT genetically has hardly started, there are hints of wide ramifications into many facets of the human condition such as mentation and aging which later will be discussed. The empirical recurrence risks for first degree relatives have been found to vary between 0 and 50% depending on the family studied. Risks appear highest in families with early onset, rapidly progressive illness, and lowest in families with affected members having onset of illness over age

70-75. These generalizations have some exceptions; many early onset, fulminating cases in families where large numbers are at risk are apparently sporatic and in all, some 30-50% of families have only one affected member.

For today's clinical practice, data from genetic studies produce insights into an extremely common and important disease, provides estimates of risks needed for counseling, and can even help greatly with diagnosis. This last is because given dementia in one member of a family, it is most likely that any other dementing member of the same family has the same disease; only one confirmed diagnosis is needed. This is a feature most important to clinical researchers because heterogenity and missdiagnosis present such difficult problems; the high risk family is a most valuable though underutilized resource.

GENERAL GENETIC AND EPIDEMIOLOGIC EVIDENCE

DEFINITION AND DIAGNOSIS OF DAT

The diagnosis of DAT requires examination of brain tissue by a competent neuropathologist who must find large numbers of neurofibrillary neurons affected by neurofibrillary tangles and senile plaques. That finding, coupled with clinical dementia, are the central features of the current operational definition of DAT. That examination of brain tissue is needed for diagnosis is a simple fact of today's medical biology that cannot be evaded no matter what difficulties it makes. And it does make difficulties, especially for research into DAT, because obtaining brain tissue for diagnosis generally requires an autopsy which is expensive (and who should pay for it?) and which entails solving problems which are logistically difficult and emotionally laden. Moreover the autopsy would ordinarily be done some years after the research had ended and the results published. So glossing over the diagnostic problem is tempting and a rationalization is available because DAT comprises the bulk of the primary dementias. Therefore, for "practical purposes", assume that all primary dementia is DAT.

The reason this temptation must be resisted is that in life, DAT cannot be distinguished from other diseases associated with the dementing syndrome. Pick's disease is the best known of the confounding diseases, but there are others, no one can say how many, which also present the same clinical picture as

DAT. Table 49-1 presents the experience on this point at Minnesota. (1) The entries in Table 49-1 consist of dementias which had been regarded by competent neurologists and psychiatrists as progressive dementias, either DAT or disease clinically indistinguishable from DAT.

(Table 49-1 here)

While the diagnostic work-ups underlying the data in Table 49-1 cannot be claimed to be uniformly perfect, they would be widely regarded as a good average of American medical practice. Nearly all of the cases depicted would have been regarded as eligible for clinical research on DAT. Yet from Table 49-1, that would be fallacious as substantial heterogenity is evident in all age ranges except the oldest. Pick's disease is an important contributor at younger ages. So are "other dementias" which consist in that series, of an atypical case of Huntington's chorea, two atypical Parkinson's, several brains with nonspecific pathological changes, a multiinfarct dementia, and a few brains which the pathologist classified as normal. Two conclusions seem warranted; classification of the progressive dementias is inadequate, and the assumption that all progressive dementia is DAT is not supported by evidence.

TABLE 49-1

Final pathologist's diagnoses in 74 cases of dementing syndrome:

Percentage of indicated diagnoses in each age interval

	Age Intervals					
Diagnoses	-39	40-49	50-59	60-69	70-79	80-
DAT	-	47	66	76	91	100
Picks disease	75	29	23	14	5	-
Other diseases	25	24	11	10	4	-

Alzheimer's neuropathology in other conditions

To add further complications, Alzheimer's pathology is not limited to DAT. The same lesions appear in the brains of aged persons who in life exhibited no decline in mentation. They appear in association with head trauma, some viral illnesses affecting brain, and with some endocrinopathies.(2) At first sight it seems self- evident that the neuropathology must be the result of a final path common to several etiologies. Yet there is an quantative difference which neuropathologists, in their practice, rely upon; DAT features an unmistakable predominence of neurons affected by Alzheimer's changes; 10-15 or more such cells per high powered field. In other conditions such as normal aging, tissue has to be searched for affected cells. The difference is sufficiently clear so pathologists regard few brains as ambiguous.

Down's syndrome and DAT

Down's syndrome provides yet another apparent exception to the specificity of Alzheimer's neuropathology which is most intriguing. Down's cases who survive to early mid life exhibit neuropathology which is indistinguishable from that of DAT.(3) Apparently all Downs who survive to age 30 are at risk and by age 40, virtually 100% are affected. Moreover, those Down's cases seem to suffer a rapidly progressive dementing process before dying. They loose skills such those needed to dress, and they seem increasingly apathetic, inattentive, uncharacteristically withdrawn, and less capable of meeting the demands of their environment; although the behavioral syndrome is hard to define precisely, the most reasonable interpretation is that all Down's are destined to develop DAT provided only that they live long enough.(4)

Future classifications

In addition to neuropathology, biochemistry and neuroanatomy seem likely to become part of the definition of DAT. It is evident that the enzyme choline acetyltransferase is present in lower amounts in cortex with DAT than in age-matched controls.(5,6) In future work, the biochemical lesions being defined in DAT will likely be used to aid classification. Similarily, it appears that a specific population of neurons may be preferentially affected in DAT. These are the choinergic neurons of the nucleus basalis. These large neurons project diffusely to the cortex and apparently are associaed with the senile plaques which develop there in DAT.(7) These same neurons are affected in the Parkinsonism-Dementia Complex which is endemic among the Chamorro people of Guam.(8) The disease features severe parkinsonian features as well as dementia. Neurofibrillary tangles (though not senile plaques) are found in profusion in the cortex. If neurochemical studies demonstrate a reduced activity of choline acetyltransferase, the evidence for a relationship between DAT and the Parkinson-Dementia complex would be strengenened. In fact, some victims of DAT, especially the early onset severely affected cases reported in family studies, do develop muscular rigidity which resembles that seen in Parkinson's disease. At this time the classification of the component diseases of the dementing syndrome associated with Alzheimer's neuropathology is by no means settled.

EPIDEMIOLOGY

The incidence of DAT in general populations has been little studied; doing the needed research is technically and logistically most difficult. Larsson and his coworkers found in Sweden, the age specific incidences seen in Table 49-2.(9) Their study was of "senile dementia" which would seem to roughly denote older cases of DAT, although ten of the cases regarded as "definite" had an onset before age 65. Nearly all diagnoses were made on clinical grounds. Akesson, also in a Swedish population,

(Table 49-2 here)

found lower estimates at the same ages, but his study populations were small and did not include milder cases.(10)

There have been several studies of the prevelance of DAT, but these too, have been severely limited by problems of diagnosis and acertainment of all ill persons in a defined population. How does an investigator disc~~y~~over all mildly or moderately forgetful elderly persons in a population, when does such a person become a case of dementing syndrome, and, if dementing syndrome is present, is it DAT? Age specific rates are a special problem. Table 49-3 presents my calculation of age specific rates using data from a Swedish study.(11) Dementia was defined

(Table 49-3 here)

conservatively in the study by means of rating scales which assessed intellectual performance. Persons with acute medical illness or severe motor impairments likely to interfere with performance on tests were excluded. The population surveyed were all persons housed in homes for the aged or were in medical facilities though not acutely ill; the total population at risk was known. Other studies suggest that about about 4.9-9.2% of Swedish dements will

TABLE 49-2

Estimates of age-specific cummulative incidence of DAT in a Swedish population.

Data from Larsson et al(9)

Age	Percent developing DAT
-70	.04
-75	1.2
-80	2.5
-85	3.8

TABLE 49-3

Estimate of age-specific cummulative prevelance
of DAT in a Swedish population.
Data from Adolfsson et al(11)

Age interval	No. dementing	% of population dementing	Prevelance of DAT Cummulative %
-69	93	0.76	1.3
70-74	150	1.55	2.8
75-79	278	4.31	7.2
80-84	320	8.92	13.2
85-89	265	30.4	23.7
90-94	107	61.2	42.9
95-	26	68.4	50.9

remain at home and thus would not have been discovered by a study based on such institutions.(11,12) Adding 7% for those at home, and assuming that the proportion of DAT among dements is about 70% (13), allows calculation of the last column in Table 49-3. The data do not permit calculation of sex specific prevelance but other data to be presented suggest that sex-related differences may not be important in the incidence of DAT.

The estimates in Table 49-3, though rough, do demonstrate essential features of the distribution of DAT. There is first, a dramatic rise in frequency following the 7th decade of life. Second, so far as observations have been extended, which includes the maximum practical age ranges, there appears to be no decrease in prevelance. DAT seems to grow more frequent with advancing age. In this it resembles ischemic vascular disease and most carcinomas but is unlike most other diseases, such as Huntingtons chorea or schizophrenia or rheumatoid arthritis, which, after specific ages, decrease in frequency. We can anticipate outliving our period of maximum risk for most diseases, but not DAT.

There are no even marginally adequate studies of the epidemiology of DAT in populations or ethnic groups except north European. Likewise, studies of environmental factors in any population have produced equivicol results with one possible exception. A recent case-controled retrospective study of a cohort of U.S. veterans undergoing dementia, discovered a apparent excess of persons with a history of traumatic injury to the head among the dementing group.(14) This finding would be consistant with the presence of neurofibrilary tangles and senile plaques in "punch drunk" boxers; trauma may thus be an etiological factor in a sub-group of DAT cases.

FAMILY STUDIES

Methodology

There have been eight studies aimed at estimating the recurrence risks among relatives of relatively unselected probands who had DAT or putative DAT. More than is usual in the study of disease, methodological points are important as these studies are described and discussed.

The first problem encountered by the researcher is establishment of diagnoses for the probands and the basic choice is between using autopsy or clinical findings. Clinical diagnoses entail acceptance of a diagnostically heterogeneous proband group (cf Table 49-1);autopsied probands force reliance on retrospective clinical data to describe the course of the proband's illness. A third choice, clinical diagnosis later confirmed or not confirmed by autopsy, seems ideal at first sight; the research can begin based on a living clinic population and can retain prospective features because the probands can be followed longitudinally until they come to autopsy. In practice, this plan has proven impractical because it requires following subjects for years through hospitals and nursing homes, where most of them will die, obtaining permissions and arranging for an autopsy.

The second major problem is how to assess disease among relatives. If affected, parents will have long since died, and an autopsy diagnosis from records, though worth seeking, will be not often be found. Siblings will likely be affected at about the same time as the proband and generally will yield the highest autopsy rates. Children will usually be too young to be affected unless the family is studied longitudinally with unusual persistance. The only practical way to assess relatives of probands, is to do intensive field evaluations, usually by interview and usually focused on siblings, and supplement the results by whatever medical records can be located and reviewed. In practice, this means that available relatives must be interviewed in the field, raport established, and contact maintained over the next decades if possible. The more meticulous the examination the better, and likewise the longer the follow-up, but practical limitations force compromises. The time required to do comprehensive psychometric testing, at present the basis for diagnosis of early dementia, and the low expected yield because even in high risk families the proportion of early cases is very low, preclude diagnosis of many early cases; only well established, unmistakable cases can be counted in most projects. An especially difficult problem is diagnosis in older subjects. After age 80-85, persons who may have some loss of intellectual competence will often have known disease of some organ system other than brain, which could account for the loss. Since doing a comprehensive work up on such persons is generally impractical, and autopsies are so difficult to arrange, an age ceiling for diagnosis is effectively established. Also, in practice, many subjects will have already died, often years before, and diagnoses, if made, will have to be made retrospectively. In such cases, diagnosis will likely be impossible unless an especially comprehensive medical record or observant family members are available who witnessed the course of the illness at first hand.

In addition to establishing the presence or absense of dementia, most researchers attempt to obtain data needed to estimate other parameters of illness. One troublesome point is estimating a date of onset for the illness. Again, close and repeated questioning of family informants will usually develop a reasonable concensus at least about the time illness was definitely present but more subtle distinctions are problematic. Medical records, if available, best date the onset, and other features of the clinical course.

The studies

Each of the eight studies of a series of families has special features which now will be described. Each study will be denoted in subsequent text by its geographical orign or, in the case of Swedish studies, of which there have been three, by the name of the major author.

Sjögren(15): The first major family study was conducted by the late Torsten Sjögren and his colleagues in Sweden. There were 18 probands with histologically verified Alzheimer's disease whose illness began by age 65. Another group of probands were clinically diagnosed as "Pick-Alzheimer syndrome" and still another group of 18 probands had histologically verified Pick's disease. Even in this relatively youthful group of probands, finding Picks to be as frequent as Alzheimer's is most surprizing and suggests a concentration of cases based on ethnicity. The follow-up, which required vigorous field work, consisted of interviews and obtaining parish and medical records. Among the families of the 18 probands with proven DAT, there were 4 secondary cases in three families, all deceased but not autopsied; a parent from each family and one sibling. Few relatives were at risk, especially when corrections are made for younger subjects who had been exposed to little or no risk, and with so few of those at risk affected, conclusive results could not be expected. Nevertheless, the Sjogren study broke new ground and has been a model ever since.

Switzerland(16): A study from Bel-Air hospital in Switzerland started with a series of autopsies. The research then consisted of finding relatives of this proband group among other patients who had attended Bel-Air clinics or hospitals. In some cases, the hospital records contained accounts of illness in other family members, which were used to establish diagnoses, but apparently no field investigations were done. Among 87 families with Alzheimer's disease, eight families had secondary cases; seven siblings, two children, and two parents had Alzheimer's disease to which should be added one sibling, one child, and four parents with senile dementia. Among the relatives of 229 probands who had senile dementia, secondary cases were found in eight families; 18 siblings, 10 children, and six parents had DAT, six of these relatives had a dementing process begining before age 65 (Alzhemier's disease), and 28 after age 65 (senile dementia). Risks to relatives for recurrent DAT were estimated at 10.6% for parents and 9.5% for sibs.

Larsson(9): Another Swedish study extended the Sjögren study by focusing on probands with senile dementia. (Though 12 probands had an onset of illness before age 65 and thus actually had Alzheimer's disease.) The 377 probands were patients in Swedish hospitals; nearly all diagnoses were clinical. Families of spouses were used as controls. The project was unprecidented in numbers of subjects and in the use of controls. Because of these features, age-specific rates for a general population and for relatives of probands could be estimated and these will provide a basis for comparison with subsequent studies. The results can be seen in Table 49-4.
At each age interval, the first degree relatives of the demented probands had about five times the risk of the general population. Field investigations of the families were intensive, though the investigators regarded their information as incomplete for about one third of their families.

Akesson(10): Yet another Swedish study aimed at evaluation of all persons living in a geographically isolated area provides comperable data. The numbers were smaller but the intensity of the follow-up of families may have been more complete than that of Larsson. The risks discovered were close to those in table 49-4.

Minnesota(17.18): A study based on 126 probands who died in Minnesota State Hospitals and had autopsies upon which to base diagnoses, has been proceding for 10 years. Probands include all 35 cases in the autopsy series age 65 or less at onset of illness plus the next 90 cases out of the 210 over age 65. Relatives are evaluated by intensive field investigations. Among 2204 relatives, 81 secondary cases of dementia have been discovered. In addition to estimation of recurrence risks, a main aim of the study is to study families longitudinally so as to define a cadre of subjects at high risk who can be followed through their illness.

Scotland(19): This study is also based on probands who had come to autopsy, 74 of them, all age 65 or less. The follow-up was done through records only. Probably as a result, there was a loss of 123 of the 595 first degree relatives of probands, or 20.7%. Unfortunately, sibs who generally yield the most stable estimates of recurrence, accounted for nearly all of the lost subjects.

North Carolina(20): This study, from the clinics of Duke University, started with probands with the dementing syndrome whose specific diagnoses were unproven. However, the 65 probands, all age 70 or less, were thououghly studied and the follow-up of the 1278 relatives was comprehensive. 22 dementing cases were found in 17 families. Spouse family controls were used.

Baltimore(21): Finally, Johns Hopkins researchers have been conducting an ongoing study based on their clinic population. The probands are 62 white females, again with clinical diagnoses only. Among 260 first degree relatives over age 60, 22 were demented compared to 4 of 157 controls. A main aim of the study has been to discover clinical features which may distinguish familial from non-familial DAT.

Reports of single families: The reports of individual pedigrees which have appeared are much too highly selected to be used in statistical analyses. However, some data of a descriptive nature is useful. The pattern of inheritance in these families has been uniformly consistant with autosomal dominance and, most often, inconsistant with any form or recessive or sex-linked transmssion. The onset tends to be relatively young, in the 4th and 5th decades, and the progression of the disease, rapid. Many such families exhibit prominent neuromuscular disturbances, mainly extrapyrimidal in nature. These reports have been thoroughly reviewed.(22,23)

Results of family studies

Recurrence risks: The family studies have developed reasonably consistant recurrence risks for parents, siblings, and second degree relatives. Age-specific incidence rates for sibs from the studies which numbers at risk and methods used to study those at risk suggest stability, can be seen in Table 49-4. Parents exhibit about the same pattern. No risks are now available for

(Table 49-4 here)

children because no group of children of probands have been followed into the ages at risk. Results vary among studies but the differences seem reasonably well explained by diferences in sampling. For example, the North Carolina study found the highest risks, but all of their probands were under age 70 at the onset of illness and, as will be shown, relatives of younger probands have higher risks. Åkesson's probands, though older, were especially severely affected while Larsson's, also older, included all ranges of severity. Given the vagaries of diagnosis and sampling, especially the age distribution of probands, it would seem

TABLE 49.4

Age specific risks for DAT in sibs of probands

Percentages, not cummulative

	Investigation				
Age interval	Åkesson*	Larsson	Minnesota	North Carolina**	Controls***
-54				1.0	
55-59			1.0	1.5	
60-64	3.5		1.2	3.5	
65-69	3.5		4.1	1.0	0.4
70-74	5.3	1.6	4.0	7.0	0.8
75-79	5.3	3.4	4.3		1.3
80-84	7.6	3.6	4.2		1.3
85-	7.6	7.1	3.2		

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*Interpolated from graphed data

**Five year intervals averaged from published 10 year intervals

***data from Larsson

that family studies are generally recognizing and recording the same phenomona.

Figure 49-1 depicts the latest updating (December, 1983) of the cumulative age specific risks from the ongoing Minnesota study. These data lead to four conclusions on which there is general agreement:

(Figure 49-1 here)

1. Risks must be cited as age -specific. It makes a tremendous practical difference if a given risk is applicable at age 60 or age 80.
2. In Figure 49-1, the cumulative risks generally appear as straight lines suggesting a constant risk through each age interval. In the Minnesota study, that risk is 4%-5% for each 5 years of life after age 60.
3. The risks for a general population, while of comperable magnitude for each 5 years of life, become appreciable only after age 80. Thus being a first degree relative of a person with DAT has the apparent effect of shifting the risk to younger ages or to the left on Figure 49-1.
4. Risks are increased by if other family members are affected. This is most marked in the Larsson and Minnesota studies but was also found by the North Carolina group. Such an increase is consistant with

experience in many common diseases and is a recognized indicator of severity. Figure 49-2 depicts the risk for sibs found in the updated Minnesota study. If a parent was affected, the risk to the sibs of probands is increased dramatically.

(Figure 49-2 here)

Genetic theory and the findings in several common disorders associated with genes suggest that in DAT, risks to relatives might also be increased by age at onset of illness; the earlier the age at onset, the greater the risk to relatives. This effect, which is also associated with increased severity of illness, is predicted by genetic theory and by findings from many common diseases. However, only one of the family studies, the Minnesota Study, had probands in sufficient numbers over a wide enough range of ages at onset. If the Minnesota families are divided according to the age at onset of the proband through whom the family was located, the relatives of probands under age 70 have significantly higher risks than those 70 and older. The effect on risks to sibs can be seen in Figure 49-2. Neither Larsson's material, which had few young probands, nor the North Carolina material which had no probands over age 70 can be used to confirm the Minnesota finding. No one study or combination of studies has enough probands with early onsets to permit making finer divisions, say into decades, by onset of proband's disease. Although, highly selected for severe dramatic illness, the individual family pedigrees which have been reported exhibit a strong association between early onset and increased risk to relatives.

In the Minnesota study, increased risk to relatives associated with early onset of illness and with illness in a proband's parent was clearly additive. In Figure 49-2, that effect can be seen; siblings of probands whose onset was less than ae 70, and who had an affected parent, had a risk greatly increased over other groups of sibs.

All studies have found a large proportion of probands to be sporatic cases with no affected relatives. The Minnesota series found 1/3 of families with secondary cases, North Carolina in 17 of 68 families, and Larsson found about the same proportion or a little greater one depending on how his uncertain diagnoses are counted. The families without secondary cases tend to be located through older probands (> age 70), which as will be seen below (Proband Effect), makes interpretation of the finding ambiguous.

Sex: Family studies suggest that there is no sex effect on recurrence risks or severity of illness. While it is true that most series of cases from clinics contain as excess of females over males, this is likely because artifacts of ascertainment favor over-inclusion of females. Females tend to be younger than their husbands and to live longer. Therefore, an ill male is more likely to have a spouse available to care for him and less likely to be hospitalized and hence at risk for inclusion in a family study. The crucial finding in family studies is that secondary cases among relatives of probands contain males and females in proportions not significantly different from those found in the general population in the ages at risk. This finding is supported by the Minnesota study which discovered no significant difference between males and females in age at onset or duration of illness. If a sex effect were present, the sex more severely affected would have, on average, an earlier onset and more rapidly progressive disease.

Proband effect: A striking finding from family studies is that probands are much younger that secondary cases from the same family. In the Swedish and the North Carolina studies, the difference was roughly 10 years, in the Minnesota study it was 5 years. This is not unexpected in direction because probands tend to have more dramatic, more severe illnesses, which would, on average, begin at younger ages and which would increase the liklihood that the affected person would become involved with medical facilities and hence be at risk for inclusion in a study. But the magnitude of the effect, 5-10 years, seems large. Moreover, it is quite consistant among families. In Minnesota, only 5 probands had an onset of illness at an age older than an affected relative; the North Carolina study found 2 probands, out of 23, younger at onset than their affected relative. This finding has practical applications. First, some methods of genetic analysis assume a similar age distribution for probands and secondary cases. Obviously, this assumption will lead to faulty analyses if applied to DAT (c.f. Whalley et al (19)). Second, the finding has far reaching implications for genetic counseling. If the expected age for secondary cases in a family is 5-10 years older than the case that brought the family to the counselor, the practical consequences may be decisive. An immediate question is whether a proband is the practical equivalent of the family member whose illness leads to contact with a counselor. At Minnesota, we have been compiling the relevant data from our clinics. In 14 counseling cases where the index case had a progressive dementia, other affected family members whose age at onset could be estimated, averaged 6.8 years older than the index case.

The evidence is sparse, but in practice, we adjust the age-specific risks according to that experience.

Birth order: A finding that persons who develop DAT are born to older mothers (24) has not been confirmed by other studies (19,20). Maternal (and Paternal) age at birth of persons destined to develop DAT are indistinguishable from their siblings or other control populations.

Diagnoses: Finally, family studies are applicable to diagnoses in several ways. So far, all autopsies of two or more persons from the same family who in life exhibited a clinical dementia, have been concordant; a diagnosis in one family memeber predicts a diagnosis in another family member with perfect accuracy. Zerbin-Rudin has reviewed this data up to 1967(25); in Minnesota, experience with 27 pairs of relatives has yielded the same perfect concordance, and I know of no reports of exceptions. Those data suggest that tissue pathology does in fact delimit specific diseases. They also suggest that high risk families, where a diagnosis has been established for one family member, are especially valuable for research purposes, and further, that as treatments and more refined prognoses become available, it will be most important that members of families at high risk know which of the dementing illnesses have afflicted their relatives.

Associated disorders

DAT has been suspected to be one major facet of a complex of diseases. Its issue pathology is shared by other process such as aging and the Parkinson-dementia complex as has been described. Relatives of DAT probands may also be at increased risk for other diseases. This possibility is far from adequately explored; the evidence available amounts to strong hints at best. One general feature seems established: if there are disorders associated with DAT, it is the members of severely affected families who are at increased risk, just as those same relatives are at greater risk for DAT itself.

Downs syndrome: A relationship between Downs and DAT clearly exists but its extent and significance are not yet clear. As described earlier, apparently all Downs who survive long enough will develop DAT; moreover, nearly all of those affected will develop the disease during their fourth decade of life. Whether or not this risk is applicable to translocation Downs is unclear, but it certainly is applicable to trisomy Downs. This suggests that a gene product from chromosome 21 is involved in the etiology of DAT, although the gene itself may not be abnormal. In Downs, 50% more of normal gene products from chromosome 21 are present; one such product could be associated with DAT. This seems a likely hypothesis if one accepts the 100% frequency of DAT in Downs and agrees that all Downs developing DAT are unlikely to be born with the same defective gene associated with DAT in non-Downs cases. If DAT in non-Downs is associated with the same gene product, its presence in a higher dose could be due to faulty regulation of a normal gene on chromosome 21, probably from a defective gene on another chromosome. Other possible mechanisms could be postulated.

A further possibility is suggested by the finding of an apparent excess of Downs cases among relatives of DAT probands in the Minnesota and in the North Carolina studies. (In Minnesota 11 of 3044 at risk and in North Carolina, 4 trisomic Downs among 1125 at risk plus 6 15-21 translocation Downs in one family.) No other study that was large enough looked for Downs cases. In both positive studies, the Down's cases appeared among relatives of probands who had early onset of illness and who were mainly members of the generation of grandchildren of probands. Whether or not persons in the generation of children of probands will develop DAT and establish a closer genealogical connection between Downs and DAT is unknown.

Malignacy: In the Minnesota study there was an apparent excess of malignacy involving lymphatic tissues.

This finding was not confirmed by either the North Carolina or Scottish study although in the latter study, the loss to follow-up of so many siblings of probands detracts from the significance of the negative result. As the follow up has continued in Minnesota, it now appears that the excess observed is attributable to families which have heavy loadings of cancer as well as DAT. In one family, there were 4 cases of DAT, 5 of chronic lymphocytic leukemia (one of those in a DAT case), two lymphosarcomas and one multiple myeloma among 15 persons who lived to at least age 10. In another family there were 5 cases of DAT, 7 leukemias and one multiple myeloma among 64 at risk; and in a third family, consisting of one sibship of 7, there were 3 DAT, and 2 lymphomas. These families would be extremely rare in any population. That they were found through probands with DAT suggests an association.

Other diseases: The North Carolina study found a suspiciously high frequency (16.9%) of thyroid disease among female probands in their study and (11.8%) among their sisters. Comperable rates of 3.3-7% were noted in studies of unselected populatons, which was comperable to the 6.3% found among sisters of spouses of the North Carolina probands. The disorders included Graves disease, hyperthyroidism and thyroiditis. This finding partly echos one from the Minnesota study. Among the relatives of younger probands (<65 years), those comparable in age to the North Carolina probands, an excess of disorders often classified among the autoimmune group of diseases. Thyroid disorders contributed importantly to that excess.(27)

GENETIC PATHOPHYSIOLOGY

While there is a developing biochemistry of DAT, there cannot yet be said to be a biochemical genetics. What has been demonstrated in brain tissue are differences in transmitter-specific markers in DAT compared to controls; chiefly involved are the forebrain cholinergic systems and the noradrenergic and somatostatinergic systems of the telencephalon. Degenerating nerve cells leave behind the plaques and tangles which delimit DAT, appear to be members of a highly specific population of cholinergic neurons tightly linked to abnormalities in transmitter metabolism. These finding have been the subject of several reviews. (5,6,28)

DNA repair

A finding which may be closer to gene action is a possible defict in DNA repair in cultured lymphocytes from DNA victims from families where at least one diagnosis of DAT had been proven by autopsy. Spouse or other age matched controls were used. The dividing cells were stressed with bleomycin sulfate, x-ray, 4-nitro-1-quinoline oxide, and mitomycin C. Bleomycin was associated with increased chromosome breakage, delay in the cell delay in the cell cycle, and a decrease in the mitotic index; x-ray treatment produced equivicol results and the other treatments had no significant effect. That pattern of hypersensitivity to mutagins is consistant with base excision repair or strand breakage rejoining deficit, or both, and is not compatable with other deficits in repair.(29)

Other recent studies are also suggestive of defective DNA repair in DAT. One study found increased premature centromere division in females with dementing syndrome.(30). Another group using viral transformed cell lines, reported decreased cell viability after x-ray treatment, but not after ultraviolet radiation.(31) This pattern of results is also consistant with a deficit in nucleotide excision repair.

Aneuploidy

A finding perhaps closely related to DNA repair has been the demonstration, albeit an inconsistant demonstration, of increased numbers of of aneuploid cells in lymphocyte cultures from clinically diagnosed DAT cases. (32,33) The positive findings consisted of increased aneuploidy involving mainly autosomal chromosomes in female DAT cases. No one chromosome seemed affected more than any other.

HETEROGENITY

The consolidation of Alzheimer's disease and senile dementia into the one entity, DAT, has been generally accepted despite the fact that those accepting it fully expect that several genetic variants, and most likely environmental phenocopies, underly DAT phenotypes. It is just that today, insufficient evidence supports subdivision.

The Baltimore group has been actively seeking evidence to warrant subdivision into familial and non-familial disease in the form of clinical criteria which would effect valid separation of cases with and without dementia in relatives. They have proposed that language disorder or apraxia distinguishes familial cases and have published some supporting evidence.(21) The North Carolina group could not confirm this result.(20)

In seeking evidence supporting subdivision, there is clearly a risk of confounding severity of illness with the familial non-familial distinction; the language disordered cases may have more severe illness and hence more affected relatives; or the proband effect described above may disguise familial cases; if secondary cases are 5-10 years older, some will die of competing causes of death before manifesting DAT and the older the proband, the greater the liklihood of wrongly assigning the case into a non-familial or sporadic category. Nevertheless, that DAT is sporatic in some large proportion of families seems evident, and search for criteria which would delimit those families is entirely logical.

COUNSELING

The basic data needed for estimating genetic risks for relatives is available in Figures 49-1 and 49-2. The counselor must decide what type of family is presenting and find the appropriate graph. Until specific risks for children are available, sibs offer the best approximation. In most cases the estimated risk will be small, and, especially when its age-specific nature is explained, most counselees will be relieved and reassured. However, there are families subject to appreciable risk who will wish to plan in the light of data. These are the families with early onset of illness, or more than one relative affected, or both early onset and more than one relative affected, as depicted in Figure 49-2.

The counselor will have to find some solution to two main problems before estimating risks for such families. The first of these is diagnosis. As is apparent in Table 49-1, early onset cases, those which imply appreciable genetic risk, cannot be assumed to be DAT. While there is some genetic risk data applicable to Picks disease, there is little or none applicable to other causes of early onset dementing syndromes.(34) Therefore, the first task is to establish a diagnosis. This can be done in some cases by obtaining medical records on all deceased family members who may have had a clinical dementia. Death certificates can be useful because they usually cite medical facilities or physicians who cared for the deceased person and whether or not an autposy was done. Certificates can be obtained provided that the correct name of the deceased and at least the State in which death occurred are known exactly, and the year of death is known at least approximately. Following leads from the certificate, records can be requested. Obtaining diagnoses in this way requires clerical work which counselees can manage and which helps involve them actively in the process. If no diagnosis can be obtained, I do not estimate risks for counselees. This is made more palatable to many counselees because they often are first made aware of the problem and seek advice because of newly diagnosed illness in a relative. Estimation of risks can wait for the results of an autopsy.

The second problem to be solved is correction of risk because of age attained. In practice, this correction is applicable in only a small number of cases with onset mostly in the late 30's to early 50's, usually from families with an apparent autosomal dominant transmission. Table 49-5 presents the age at onset for DAT as observed in

(Table 49-5 here)

Minnesota. In the absense of more specific data, Table 49-5 can be used to estimate the proportion of the risk period which has passed, thus allowing estimation of the remaining risk. Because of the wide variation in DAT families depending on severity, this is not as satisfactory as determining which family type is present and estimating the risk passed from Figures 49-1 and 49-2.

GENETICS AND THERAPY

A potential use of genetic information is to guide therapy. While today there are no~~t~~ ~~now~~ effective treatments, there is reason to expect that these will be developed over the next decades. This provides excellent reason for relatives of DAT cases to learn about the disease and about their family. Because, a diagnosis in one family member leads to a highly likely diagnosis in any other family member who develops a dementia, a rationale

TABLE 49-5

Age at onset of DAT:

Minnesota series of 239 cases

Age interval	Percent with onset	Cumulative percent
-44	2	2
45-49	3	5
50-54	5	10
55-59	7	17
60-64	14	31
65-69	19	50
70-74	17	67
75-79	16	83
80-84	12	95
85-	5	100

for presumptive diagnosis and prompt treatment of a known specific disease may be immediatly available to the treating physician; the time saved could hardly fail to be significant. The liklihood of heterogenity and that different variants of the disease will respond to different treatments, gives families excellent reason to keep in close contact with developments in medicine and to develop comprehensive, detailed medical pedigrees.

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Mr. **Gore**. Thank you very much. We are going to save our questions until we hear Dr. Manuelides.

Dr. Manuelides is professor of pathology and neurology at Yale. We are delighted to have you here. Thank you very much for coming, and please proceed.

STATEMENT OF ELIAS EMMANUEL MANUELIDES, M.D., PROFESSOR OF PATHOLOGY AND NEUROLOGY, YALE UNIVERSITY SCHOOL OF MEDICINE, NEW HAVEN, CT

Dr. **Manuelides**. Many thanks for giving me the opportunity to speak before your committee.

As you heard yesterday and today from several witnesses, the causes of Alzheimer's disease are unknown. It is a mystery up to the present time, the etiology of this condition or these conditions. I shall restrict my remarks to the possibility that a slow virus is implicated in Alzheimer's disease, and an infectious etiology may play a role in this condition.

I would like to start by defining that the slow virus cause diseases which appear several years or decades after exposure to the infectious agent. Now, example of these diseases are some virus dementia which are proven beyond doubt they are caused by an infectious agent, and such examples are kuru, which is a condition described in the South Pacific; Creutzfeldt-Jakob disease of men; and scrapie, a condition occurring in sheep.

May I have my first slide. I am a neuropathologist, and without my slides I cannot illustrate my thoughts.

Mr. **Gore**. You have the names of these diseases written out?

Dr. **Manuelides**. Yes, I have written the names out.

As you can see in the first slide, these are transmissible conditions. Kuru and Creutzfeldt-Jakob disease in men are characterized by dementia, and it is proven beyond any doubt that they are infectious conditions.

May I have the second slide, please. Now, the suggestion arises, are there any similarities or any overlapping between the clinical changes in Alzheimer's disease and the clinical changes observed in this virus dementia. As you can see here, the onset in both Alzheimer's and Creutzfeldt-Jakob disease is roughly the same. All conditions, Alzheimer's as well as the other diseases cause dementia. Myoclonus is a twisting of the muscles, a prominent sign in Creutzfeldt-Jakob disease, occurs also in Alzheimer's disease.

Brain wave changes may be very similar in both conditions.

Now about the familial incidence. We have a familiar incidence of 15 percent in Alzheimer's disease, and we have a familial incidence also of some 15 percent in the virus dementia. Now, there is a very fascinating observation that you may find some families where both conditions occur. Some members have Alzheimer's disease, and other members have a virus dementia. Furthermore, there are a few instances where you can see in one and the same person the changes of Alzheimer's diseases and the changes of a virus dementia. The incubation period is very long in the virus dementia. In Alzheimer's disease it is totally unknown. These are the clinical similarities.

I would like to point out now the pathological similarities, what you see in the brain in both conditions, in virus dementias and Alzheimer's disease.

May I have the next slide, please? At first to refresh your memory. The lesions occur in the cardinal lesions in Alzheimer's disease. We have these deposits, these senile plaques here in Alzheimer's disease.

May I have the next one, please? And the neurofibrillary tangles.

May I have the next slide, please? In the virus dementias, what we have are these spongy changes in the brain. For this group of diseases, it is called spongiform virus encephalopathies. These are the spongy changes. It is from one of our patients.

Next, please. Here is the electron microscope, and we see the spongy changes.

Next, please. Here is a structure recently discovered in this virus dementia, the SAF or scraple associated fibrils, which are associated with high infectivity. It may be the virus. It may be a pathological product. Up to the present time, it is found only in virus dementia and has not been seen in Alzheimer's disease.

As you can see, the clinical similarities are really overlapping, and in most instances you need an autopsy before you clarify the situation. I can give you examples from our own activity in Yale, where we do not know up to the moment of autopsy whether a person has a virus dementia or Alzheimer's disease.

May I have the next slide, please? Now you see the similarities, the pathological similarities. Spongy changes, the hallmark of Creutzfeldt-Jakob disease may occur in Alzheimer's disease. The tangles seen in Alzheimer's disease are present also in virus dementias. The plaques are the most fascinating, the hallmark lesion in Alzheimer's disease, seen also in virus dementia. As a matter of fact, one of the recent advantages of research is that you can produce plaques experimentally with viruses.

Next, please. You heard this morning from my colleagues, Peter Davies and Charles Marotta, that there are some neurochemical changes in Alzheimer's disease. You heard about the decrease of choline acetyltransferase in enzymes. In some strains of mice, you can find the same decrease in virus dementia. So the situation arises where the increase of choline acetyltransferase is the cause of the result of this disease, and there are also immunological similarities between these two conditions.

It is obviously very stimulating to do research work and try to demonstrate whether there is an infectious etiology in Alzheimer's disease. We try in our laboratory. We try in our National Institutes of Health. Up to the present time, we have not been successful in transmitting Alzheimer's disease either to monkeys or to convenient laboratory animals like guinea pigs, hamsters, and mice. But that may be because we investigated the wrong end of the disease. All our transmission experiments are from autopsy material, from end stages of Alzheimer's disease. We never have tried to investigate an infectious etiology at very early stages of the disease, when the individuals have no other symptoms or a loss of memory for recent events. It is very difficult to obtain such material, but not impossible.

So we move along these lines, my laboratory and several others' laboratories, to try to demonstrate the infectious etiology of Alzheimer's disease. As I mentioned to you, it is really a major achievement that senile plaques can be produced in convenient laboratory animals experimentally with an infectious agent.

Now also we utilize the models derived from this conditions, Alzheimer's, from these virus dementias like Creutzfeldt-Jakob's disease and scrapie in sheep in order to study the step-by-step evolution of the condition from the beginning to the bitter end. We do not have, unfortunately, any animal models derived directly from Alzheimer's disease, but we have these models that can be very useful because we can terminate our experiments any time at our convenience, we can influence the pathogenesis, the neurochemical changes by various means. So we have a very powerful tool in our hands by using this model, not only to study virus dementias, but to study also dementias from unknown etiology like Alzheimer's disease.

Thank you very much for giving me the opportunity to talk to you. I shall be open for any questions you may have about transmission experiments, about autopsies, and so on.

[The prepared statement of Dr. Manuelidis follows:]

ON THE POSSIBLE INFECTIOUS ETIOLOGY OF ALZHEIMER'S DISEASE AND THE RELATIONSHIP OF THIS CONDITION TO TRANSMISSIBLE DEMENTIAS

I, Elias E. Manuelidis, M.D., Professor of Neuropathology and Neurology, Yale University School of Medicine:

At the present time any rigid classification of dementias and more specifically of Alzheimer's disease is inappropriate because the causes of these diseases are not entirely understood and their pathogenesis is obscure. It is agreed upon that one of the most common dementias affecting individuals in the United States is Alzheimer's disease. A very prominent medical scientist, Dr. Lewis Thomas, called the group of dementias "the disease of the century". Dementias are the third or fourth most common group of diseases affecting individuals sixty-five years of age or older. Alzheimer's disease accounts for over 50% of the cases of dementia, and according to some statistics as many as 4,000,000 Americans, 65 or older, suffer from Alzheimer's disease. With the rapid increase of our geriatric population, it is projected that there will be a rapid rise of Alzheimer's disease. Accordingly, the Federal funds for long term care will increase from the present level of $12,000,000,000 to $43,000,000,000 by the year 1990. Alzheimer's disease accounts and will account for up to 50% of this expenditure.

Alzheimer's disease is first detected clinically by loss of memory for recent events and mild intellectual deterioration. With the passing of time the cognitive impairment progresses and is associated with personality changes, irritability, and severe disorientation as to time and space. Ultimately these patients lose all intellectual abilities and reach a stage of complete vegetation. The victims of this condition do not die from Alzheimer's disease but from other intercurrent diseases, such as pneumonias, bed sores, sepsis etc. Therefore, death certificates do not accurately reflect the true incidence of Alzheimer's disease. The progression and duration of the illness varies very considerably. The course of the disease is more rapid in younger patients than in older patients and in some instances the disease may last for years even decades.

Alzheimer's disease is a debilitating condition for both the patients and their families. It is worse than cancer. A cancer victim recognizes his surroundings, can communicate with family and friends, and is cognizant up to the last days of his life of the tender care of the community. An Alzheimer victim is intellectually and emotionally mute. An impenetrable concrete wall has descended between him and the world. At the present time there is no prevention and no treatment of this disease.

The clinical diagnosis of Alzheimer's disease especially in the early stages is difficult, and only autopsy and a careful post-mortem examination of the brain, can clarify the situation.

Alzheimer's disease is an organic brain disease and the post-mortem findings are characterized by 1) senile or neuritic "plaques" which are accumulations or deposits of protein and other cellular debris in the brain. 2) neurofibrillary "tangles" which constitute accumulations of fiber like proteins inside the nerve cells and 3) granulovacuolar degeneration of the nerve cells in which we have deposit of material of uncertain origin inside the nerve cells. In addition, there is loss of nerve cells and gliosis (which is an increase number of supporting cells occurring in the central nervous system). The loss of nerve cells and gliosis is more prominent in some regions of the brain than in others. I would like to emphasize that one of the most intriguing lesions occurring in Alzheimer's disease are the "plaques".

The cause or causes of this most important disease are unknown. Some of the possible causes are the following:

1) Loss of nerve cells in the brain in specific regions (nuclear basilis of Meynert, locus ceruleus). This loss is similar to the loss of nerve cells in specific regions of the brain in Parkinson's disease. The reason for this loss is unknown and it may very well be that the loss of nerve cells is the result, rather than the cause, of Alzheimer's disease.

2) Condensation and deposition of fibrillar proteins in nerve cells which interfere with the normal function of these neuronal elements.

3) Aluminum intoxications.

4) An immune disorder has been suggested as a possible cause of Alzheimer's disease.

5) Genetic causes in Alzheimer's disease.

6) An atypical infectious agent is thought to be implicated in Alzheimer's disease ("slow virus").

I shall restrict my remarks to the possible infectious etiology of Alzheimer's disease and the possibility that this condition represents a "slow virus infection" of the central nervous system.

As the name implies some viruses produce diseases several years after exposure to the infectious agent. The period from the time of exposure to the time of the appearance of the first clinical signs and symptoms is called the incubation period. The incubation period in slow virus diseases is very long and in some instances, can cover several years. In the interim there are no clinical manifestations of the disease. Examples of slow viruses disease are kuru, a condition described in New Guinea, and Creutzfeldt-Jakob disease which occurs throughout the world; both diseases affect humans. A similar slow virus disease is scrapie which is found in sheep. These three diseases are caused by unconventional viruses, unconventional due to the unusual biological and physical chemical properties of the infectious agent. In both kuru and Creutzfeldt-Jakob disease one of the characteristic signs of the disease is the occurrence of dementia. One important lesion in the brain of patients of kuru and Creutzfeldt-Jakob disease is the presence spongy changes (vacuoles in the brain). Kuru, Creutzfeldt-Jakob disease, and scrapie are grouped together as spongiform virus encephalopathies or transmissible encephalopathies. As stated the infectious etiology of these conditions is demonstrated beyond doubt and all three have been transmitted experimentally to a variety of animals. We shall try to demonstrate the relationship and similarities of Alzheimer's disease to Creutzfeldt-Jakob disease, a transmissible dementia. The similarities between both are as follows:

Clinical similarities. These two conditions may be so similar that only autopsy can clarify the situation. The onset of the disease is roughly in similar in both conditions. Dementia is present in both conditions. Shock-like muscular contractions affecting part or the whole of the muscle (myoclonus) which is a prominent sign in Creutzfeldt-Jakob disease and is also present in Alzheimer disease. Brain wave abnormalities (electroencephalograhic changes) occur in both conditions. Familial incidence or familial cases constitute roughly 15% of both Alzheimer's disease and Creutzfeldt-Jakob disease. There is joint occurrence of Alzheimer's disease and Creutzfeldt-Jakob disease in single families; some members of a family can be afflicted by

Creutzfeldt-Jakob disease and other members have Alzheimer's disease. Joint occurrence may also occur, although rarely, in one individual, and cases having both the characteristic post-mortem changes of Alzheimer's disease and Creutzfeldt-Jakob disease have been reported. Pathological changes in the brain also overlap in Alzheimer's disease and Creutzfeldt-Jakob disease. Spongy changes thought to be characteristic of Creutzfeldt-Jakob disease have been described in Alzheimer's disease and vice versa. The plaques which constitute the hallmark of Alzheimer's disease have been seen also in Creutzfeldt-Jakob disease. Plaques occur in almost 100% of the cases of Alzheimer's disease, and occur in approximately 9% of Creutzfeldt-Jakob disease cases. Indeed, in a subgroup of Creutzfeldt-Jakob disease (Gerstmann's Streussler disease), the incidence of plaques is considerably higher. Plaques in this variant of Creutzfeldt-Jakob disease are always present and are found in large numbers. It should be emphasized that plaques have been experimentally produced in slow virus disease (both in Creutzfeldt-Jakob disease and in scrapie) in experimental mice. Neurochemical similiarities. A marked decrease of choline-acetyltransferase has been reported in Alzheimer's disease; a decrease of the same enzyme has been reported in some spongiform virus encephalopathies (scrapie). Immunological similarities. Serum neurofilament autoantibodies have been reported in 50% of the cases of Creutzfeldt-Jakob disease and in 25% of the cases of Alzheimer's disease.

The similarities between Creutzfeldt-Jakob disease and Alzheimer's disease and the quoted reports from the existing bibliography makes the search for an infectious etiology in Alzheimer's disease a most stimulating and attractive undertaking. The existing similarities should serve as a impetus of future experimental work in Alzheimer's disease. Unfortunately, up to the present time all attempts to transmit Alzheimer's disease to experimental animals have failed. The additional report from our National Institutes of Health of successful transmission of some cases of familial Alzheimer's disease has been retracted. We try in Yale to transmit classical Alzheimer's to convenient laboratory animals; however, our findings have been entirely negative thus far. The lack of successful transmissions to animals in Alzheimer's disease may be due to the fact that we were looking at the wrong end of the disease. Indeed, animal transmissions were attempted only with material at terminal stages of Alzheimer's disease. As a matter of fact, most information available to us at the present time about Alzheimer's disease is by careful examination at the end stages of this condition. Yet it is a very well known fact from general virology that even at end stages of conventional viral encephalitides, the amount of the virus may be so low as not to be detectable in transmission experiments. It may very well be that this is the reason why we have not yet been successful in transmitting the disease. Maybe if we have a look at the other end of the disease, at the earliest possible stages of Alzheimer's disease, we could be more successful in showing an infectious etiology. A major obstacle in transmission experiments is the lack of human material derived from early stages of Alzheimer's disease.

It is a very well known fact that in animal models, an investigator is able to study a given disease from its earliest inception to the bitter end. In this way one can study in depth the mechanisms of the disease. There are no animal models available directly arising from human cases of Alzheimer's disease. However, there are very valuable and convenient animal models derived from transmissible (spongiform) encephalopathies, such as Creutzfeldt-Jakob disease and scrapie. We already have used the models developed by us of human Creutzfeldt-Jakob disease in guinea pigs, hamsters, and mice for the general study of dementias including Alzheimer's disease.

Even if no infectious agent shall be demonstrated in Alzheimer's disease, what is important and relevant is to unravel common steps and pathways in the pathogenesis of dementias of unknown etiology and transmissible dementias. The available models of transmissible encephalopathies such as scrapie and CJD, in which the step by step evolution of an unremittent disease can be followed from the beginning to the bitter end, is a powerful investigative resource. Infectious agents present in transmissible encephalopathies may be shown to trigger changes and disease mechanisms that constitute the same final pathways to all dementias. Indeed, in such models evaluation of therapeutic regimen may not be necessarily directed against the infectious agent, but may be useful at some step(s) in their common pathways.

Conclusion.

The causes of Alzheimer's disease are unknown at the present time and several working hypotheses have been advanced in search for the etiology of the debilitating disease. Available data indicate the possibility that an unknown infectious agent may be implicated in this condition, and attempts should be undertaken for the further transmission of additional human cases of Alzheimer's disease to laboratory animals; these transmission attempts should preferably be undertaken from material derived from early stages of the human disease.

Available animal models derived from transmissible dementias should be used for the study in depth of dementias in general, including those of unknown etiology such as Alzheimer's disease.

Mr. **Gore**. Thank you very much. Thank you both for your testimony. Let me ask some questions here.

Dr. Heston, you have submitted a scholarly and rather detailed statement, and we appreciate it very much. I believe you said that you found no sex difference in the prevalance of Alzheimer's disease; is that correct?

Dr. **Heston**. That is correct.

Mr. **Gore**. Yet there are some claims that more women are affected than men. Is that simply because of their greater longevity? Is that the only reason?

Dr. **Heston**. No, I think it is artifactual because of the ascertainment. Almost all such studies depend on people coming to medical clinics. Because women tend to be younger than their spouses, and they tend to live longer, the probabilities of a male having someone at home to take care of him is much higher than for a woman. This is one example of the kind of thing that can distort those estimates. I think an unbiased estimate is provided by the relatives of persons who become ill, and there are males and females who are equally affected.

Mr. **Gore**. I see. If I understood you correctly, when Alzheimer's disease occurs in a patient, particularly at a relatively young age, there is a greater likelihood that another family member will have the disease?

Dr. **Heston**. That is correct.

Mr. **Gore**. Is that also true, there is a greater likelihood that another family member will have the disease, but that family member will have it at an older age; is that correct?

Dr. **Heston**. On the average, the second person affected, becoming known to medicine, will have it an older age, almost certainly because the first person coming to notice will have a more dramatic and more flagrant and more severe illness and, therefore, will appear in medical clinics at a younger age. In our studies, it is running 5 or 6 years difference, which is of some practical importance.

Mr. **Gore**. I see. The younger the patient, the greater the risk is for the relatives?

Dr. **Heston**. Yes.

Mr. **Gore**. How would you quantify that risk? Is it still relatively small?

Dr. **Heston**. It is relatively small, except in these few very severely affected families. Most families can be reassured by the information. There are a few families—how many, I do not know, but some—who have a real problem on the order of magnitude of Huntington's disease.

Mr. **Gore**. Let me ask you to elaborate on the reassurance, because there are a lot of families who have experienced this with a relative. When scientists speak of "risk" you are sometimes in the category that is equated with being hit by a meteorite or a lightning bolt. How do we make this understandable? Is it a relatively small risk?

Dr. **Heston**. In our case, only about one-third of all families studied have any secondary case at all, anyone else in the family. So two-thirds of the families have none.

Mr. **Gore**. Zero enhanced risk?

Dr. **Heston**. Zero risk. Then the families at the very high risk, where the age of onset tends to be in the forties and fifties, are in a very small number. I think there are perhaps 5 out of 129 families that we have studied. So those families have a problem, and that would be on the order of 3 to 4 percent of all families.

Mr. **Gore**. Those families, when you say they have a problem, those are the families where a family member has had Alzheimer's in the forties or fifties?

Dr. **Heston**. Yes, and they tend to be at quite high risk.

Mr. **Gore**. When you say "quite high risk," how high above the average? How many percentage points above the average?

Dr. **Heston**. Perhaps the total risk by age 70 will be running about 40 percent in such families. Families in between those two have varying degrees of risk, but most often it is negligible compared to the other vicissitudes that life offers us.

Mr. **Gore**. The only families who really ought to be concerned about the implications for other family members, if somebody has Alzheimer's disease in the family, the only ones who ought to be concerned and maybe take special steps to keep in touch with doctors and have checkups and that kind of thing are those families where the incidence of the disease occurs in the forties or fifties?

Dr. **Heston**. Forties, fifties, maybe up to the early sixties, yes.

Mr. **Gore**. You showed that slide, which you said was the first time you had shown it, although the information had been available than before. Does that slide show a greater-than-expected association between Alzheimer's and cancer?

Dr. **Heston**. In a very few families. Overall, there is a very marginal increase, if any. But in some of these very severely affected families, there are concentrations of disease that are quite unusual. What to make of this, I do not know, but I do think it is an opportunity for intensive study of these few families.

Mr. **Gore**. Dr. Manuelidis.

Dr. **Manuelidis**. Yes. May I show you an additional slide? Just one, on what Dr. Heston stated.

We have produced from material from infected cells arising from virus dementias cancer these mice here. It is very fascinating what Dr. Heston stated. It is very fascinating information, for instance, that in Down's syndrome, we have very high instances of leukemias, lymphomas, and other cancers. It may very well be, because after all one of the concepts we have nowadays is that cancer is a slow virus disease. We have some evidence along these lines. It may very well be that there is some link between what you find in some of the slow virus dementias and what you see in some experimental mice here.

Mr. **Gore**. Dr. Manuelidis, if you are able to prove that the disease is caused by a virus, does that mean that the disease could be prevented by vaccines, for example, or could be cured by some antiviral drug?

Dr. **Manuelidis**. Yes, it is such a possibility to have such other viral conditions where treatment can eliminate a disease. We do not have, as I mentioned to you, up to the present time—I would like to emphasize—any hard core experimental evidence that Alzheimer's disease is caused by a virus, only by analogies and information about the clinical similarities and pathological similarities.

Mr. **Gore**. It is just one of many hypotheses?

Dr. **Manuelidis**. One of the many hypotheses, but it is very popular nowadays. As you know, there are other laboratories besides my own laboratory. At the National Institutes of Health, they are working very vigorously along these lines. In the Institute of Mental Retardation, in upstate New York, they are working along these lines; Stan Prusiner on the west coast works also on the possible infectious etiology in Alzheimer's disease; and people in England. So it is a very popular hypothesis nowadays. It is not very farfetched, after you see the similarities between the two conditions, and especially families where some members get Alzheimer's and others get a virus dementia.

Mr. **Gore**. You have tried to transmit the disease to experimental animals; right?

Dr. **Manuelidis**. That is correct.

Mr. **Gore**. And so far you have not been able to do that?

Dr. **Manuelidis**. So far, all negative. More experiments were undertaken at the NIH, with several cases of Alzheimer's disease, but all were negative. Now, I would like to emphasize that all of us use end-stage material. If you take, for example, myelitis, if you try to isolate a virus from a polio victim in autopsy, you are not going to be successful. You are not going to isolate any virus because it is very late. The virus has high peak in polio, for instance, in the first day of fever. So it may very well be that there is a possibility that we examine the wrong end of the disease.

Mr. **Gore**. Very good. Again, we could certainly spend a lot more time with both of you. Excuse me, I forgot Mr. Winn. Mr. Winn?

Mr. **Winn**. Thank you.

I would like to have both of you comment on this. It might be very helpful to several of us in the room. There are several in the room who have parents or spouses with children that are sick or have passed away with Alzheimer's disease. What would you gentlemen suggest to those of us involved, as far as any family testing is concerned and how far down the line would you suggest that we go. I know there are some children of an Alzheimer's parent in the room, too.

Dr. **Heston**. I should say first of all to such a family, you want to be very sure of the diagnosis, and that is generally not the case. The diagnosis is usually clinical, that is, it is made without the benefit of an autopsy. If you look at the families of highest risk—that is, the younger families or the earlier onset—you find a very high representation of Pick's disease and all kinds of other illnesses in those cases. So the first thing is to get a diagnosis.

Then I think the thing to do is to look at the configuration of families, as I have outlined, and there is more detailed information available on that. Most families will find that they are in a range where their risk is very small. There are only a few families who need to be concerned, and those families I think should get to a competent geneticist or genetic counselor who can estimate the risks fairly precisely.

Mr. **Winn**. Doctor, do you agree with that?

Dr. **Manuelidis**. I agree, and also the diagnosis. The diagnosis of Alzheimer's disease in early stages is extremely difficult. I am active in the community, in the Alzheimer's Disease and Related

Disorders Association. I suggest always to have a second opinion from a second expert. Pure depression can mimic very easily the early symptoms of Alzheimer's disease, and the diagnosis is extremely difficult sometimes.

Mr. **WINN**. All right. Let us go to a third generation of an individual who has been diagnosed and has passed away from Alzheimer's. Other than the suggestion that Dr. Heston made, there seems to be a tie with other diseases and other viruses. Would the type of doctor that you suggested, Dr. Heston, be looking for all of those?

Dr. **HESTON**. Yes, I think so. We are talking here about rare and dramatic families. The common situation is someone in a third generation——

Mr. **WINN**. I know you are talking about rare and dramatic families; you said that with your charts. But how is a family to know if they are rare and dramatic. Something has to wave the flag.

Dr. **HESTON**. They would have to go to a competent geneticist or genetic counselor. In the case you mentioned, a third generation with no intervening disease, in our experience, the risk to members of that third generation is negligible.

Mr. **WINN**. Has there been any tie between Alzheimer's and depression?

Dr. **MANUELIDIS**. No.

Dr. **HESTON**. As a cause?

Mr. **WINN**. Not necessarily as a cause. Is there any tie that people who suffer from Alzheimer's in their early stages might have been manic depressives?

Dr. **HESTON**. Yes.

Mr. **WINN**. Dr. Manuelidis said no.

Dr. **MANUELIDIS**. Yes. I do not know whether they have been manic depressive, but one of the characteristics of the early symptoms is that individuals are depressed. They are highly intelligent individuals, and suddenly they observe that something is radically wrong with them, and they get profoundly depressed. So you have the signs and symptoms of Alzheimer's disease, plus the depression because they recognize that something is radically wrong with their intellect.

Mr. **WINN**. I understand that, and that came up a little bit yesterday. But could a person or a family who has gone through several people in the family who are manic depressives, could that be a preliminary in later years to Alzheimer's?

Dr. **MANUELIDIS**. I do not think so.

Mr. **WINN**. You do not think there is any tie whatsoever?

Dr. **MANUELIDIS**. No.

Dr. **HESTON**. Nor do I.

Mr. **WINN**. In the little booklet that we have here, it refers to alcoholism. Is there a definite tie between alcoholism and Alzheimer's?

Dr. **HESTON**. I think not. Alcoholism can look a little bit like Alzheimer's disease, but no, there is no tie.

Mr. **WINN**. We had a witness yesterday say that his wife, after she had a couple of little strokes and realized that something was wrong with her, I gather from his testimony, began to drink. They then thought that maybe her problem was alcoholism, but then after they ran a series of tests that showed she was not an alcoholic and that she was an Alzheimer's patient.

Dr. **Heston**. If I may say, I think you have to pay attention to base rates. Alcoholism is extremely common. You are looking at 8 to 10 percent of the population. So it would not be surprising to see the coexistence of two diseases of this kind. In my clinical experience, persons with Alzheimer's disease do not tend to abuse drugs; if anything, they tend to go the other way and do not often become drug abusers as a result of drugs. As the brain fails, they tend, if anything, to decrease the use of alcohol. I am sure there are exceptions to that, but in general that has been my experience.

Mr. **Winn**. A report that the committee has here on background of Alzheimer's says this. I may not pronounce some of these things right, but it says "Genetic defect. There is some evidence that a genetic defect may contribute to one form of Alzheimer's disease. This form is called familial Alzheimer's disease because it occurs in families. The prevalence of a familial Alzheimer's is controversial. The estimates of familial AD range from 10 to 60 percent—" which in my opinion is quite high.

Dr. **Manuelidis**. Five-oh? Fifty or fifteen?

Mr. **Winn**. Ten to sixty. This is not my report. This came from the Congressional Research Service. It said, "The estimates of familiar AD range from 10 to 60—" sixty "—percent of all cases. The pattern of inheritance in the familial form of AD is thought to be autosomal dominant, or the gene is found to be a chromosome other than the sex chromosome and is likely to be manifest in 50 percent of the offspring."

I do not know where they got this background material. They usually get this stuff out of medical journals.

Dr. **Heston**. There is, to begin with, a certain degree of circularity there. If you define "genetic" as "familial" and "familial" as "genetic," then of course you are going to find the two related. I think as far as I am aware, I would rely on our own experience that about one-third of the cases have an affected relative. Now, you can do some statistical corrections, and you can make that up perhaps a little higher than that, but I do not see in our material any possibility of arriving at 60 percent.

Mr. **Winn**. That is quite a range, from 10 to 60.

Dr. **Heston**. We should do better, but the information is hard to get.

Dr. **Manuelidis**. I know of up to 10 to 15 percent, but I never heard the No. 60. I do not believe that is possible.

Mr. **Winn**. Thank you very much. Thank you, Mr. Chairman.

Mr. **Gore**. Thank you. Again, let me thank both of you, as members of this panel. We are very grateful for your testimony here and for the great work that you do. Again, on behalf of a lot of people, let me thank you for your dedication and your hard work.

We have one more panel, and I would like to call them to the witness table at this time. Dr. Peter Rabins, assistant professor of psychiatry at Johns Hopkins; Dr. Carl Eisdorfer at Montefiore Medical Center in the Bronx; and Mr. Glen Kirkland, with the ADRDA chapter in Maryland, the Baltimore Central Chapter. We are delighted to have all three of you here.

This is the last panel today, and the last panel of our 3 days of hearings. It has been a very interesting hearing, and I think it is appropriate that we close with this panel. The issue of care and financing is perhaps the most difficult. We are very pleased to have these three witnesses to help us in our effort to deal with it. We will begin with you, Dr. Rabins.

Without objection, your statement and those of our other witnesses will be included in full in the record at this point. I do want to note also for the record, Dr. Rabins, that you are coauthor of this book, "The 36-Hour Day," which families of Alzheimer's patients in the audience and elsewhere swear by and hand out to people. When some Congressman comes to visit one of their centers, they say, "You have to read this." This book has meant a lot to people who have reached out for some help and information, and we are delighted that you could join us today. Please proceed.

STATEMENT OF PETER V. RABINS, M.D., ASSISTANT PROFESSOR OF PSYCHIATRY, JOHNS HOPKINS HOSPITAL, BALTIMORE, MD

Dr. Rabins. Thank you, Mr. Chairman. As we have heard this morning, Alzheimer's disease and the related disorders are a group of progressive debilitating conditions caused by deterioration of the brain, but the effects of the condition go far beyond the individual and do affect family, friends, and ultimately, the community. I would like to structure my testimony around four questions that I think are important in trying to understand how it does actually affect the family.

The first question is just: What aspects of Alzheimer's disease actually affect the family? Maybe it is obvious, but the first one is the symptoms of the disease itself. I think most of us have known someone who was called senile or suffered from some form of dementia, and we are quite aware of the fact that memory is one of the cardinal problems. Indeed, in Alzheimer's disease, memory loss is usually the very first symptom. But really the symptoms go far beyond that.

Many people lose the ability to carry out everyday activities, such as eating with a knife and fork, brushing their teeth, dressing. This is not just weakness; it is an actual loss of the brain's ability to coordinate these activities. Often, a person is quite strong. They just somehow cannot carry out what to them have become quite complicated activities.

So they often develop, 3 or 4 years into the disease, difficulty communicating. That is, they cannot speak what they want to say, or they cannot really understand what is being said to them. This can be a real source of frustration, both for the ill person and for their family. In the late stages, the person can even become unable to walk—his body just forgets how to walk—and to control bodily functions, such as urinating, so they become incontinent.

In addition to these kinds of physical symptoms, there are other symptoms that we might think of more as behavior problems perhaps. Aimless wandering is one such symptom, and another one is sleep problems. Indeed, from my point of view as a clinician, helping an ill person with Alzheimer's disease sleep is often one of the most important things I do. It not only helps them function better, it helps the family have some regularity. If they know the person is sleeping at night, they do not have to worry that they are going to fall down the steps. It seems, at least, that people are sometimes more functional in the daytime if they have had a good night's sleep.

As you can imagine, dealing with these physical symptoms on a day-to-day basis can really be quite difficult. Families not only have to live with that aspect but also with the knowledge that it is a progressive condition and that things may well get worse over time. So it requires that the family be cognizant of these ever-changing physical symptoms. This kind of care can really lead to exhaustion and fatigue in the spouse or whoever is taking care of the person.

In addition to these physical symptoms that the family is dealing with on a day-to-day basis is the emotional effect that caring for a loved one who is deteriorating has on a spouse. Relationships that have developed, often over a lifetime, are suddenly changed, and that can put a tremendous strain on families. So it is not surprising that when you ask families how they are doing, they will often tell you that they are discouraged at time, they get angry, they feel embarrassed that they have even yelled at the person once or twice. They feel guilty about things. And these kinds of feelings are very commonly reported by families of persons who have Alzheimer's disease.

The disease can also cause a real major change in just the social fabric of the family. If you had a relationship with another couple and did things with them as a couple, you really cannot do that any longer. So not only are you physically exhausted at the end of the day, but often the well person has lost a lot of the social contacts or a lot of the supports they might have had earlier. I think that these kinds of physical symptoms and the emotional effects on families are two important effects.

A third important specific effects are financial issues. I know Dr. Eisdorfer is going to talk a bit more specifically about them, but things such as retiring earlier to take care of a loved one, or paying out the expenses—if you have to have someone stay with the person while you go shopping—these can be difficult. But obviously, the most devastating effect is nursing home placement. It is my belief that nursing homes are a necessity for some patients with Alzheimer's disease. The average cost now of a nursing home in the Baltimore area is $20,000 a year—and that is just for the bed.

This week, one of my patients is, unfortunately, having to go into a nursing home. His wife cannot care for him any more. He is somebody who cannot feed himself. It is going to cost her more than $3 a day extra just to the nursing home because he will have to be fed. So that adds up to $100 a month. These are very proud people. They have saved all their lives. In 2 or 3 years, their entire life savings, which they had saved for retirement, will be gone. That is certainly quite sad.

A second question that I think should be addressed is: What are families actually doing now to take care of people with Alzheimer's disease? I think it is surprising for most people to learn that. In fact, the majority of people with Alzheimer's disease are living at home. They are not in institutions. They are usually being taken care of by families. The best estimates are that about two-thirds of people with these diseases are currently being taken care of by their families.

When you look at the kinds of people who are in nursing homes and other institutions, I think it makes it even more clear that people in nursing homes tend to be sicker, tend to have no children or that have never been married. They are people who are less likely to have somebody who could take care of them at home. So I think it is important to keep that in mind.

When we talk about Alzheimer's disease, we sometimes do tend to get overwhelmed by the enormity of the problems and lack of specific treatments. But just the fact that the majority of people are committed to taking care of their loved ones in this way really destroys the myth that most Americans do abandon their ill elderly. I think that is something we should take pride in. I think it offers us a way to make a difference to these patients and families. That is, we cannot cure the disease. We may not even be able to slow it down; we probably cannot slow it down. But if we can work with the families and help them cope better, help decrease some of the symptoms, I think we can make a very positive difference. I think those of us who care for these families take pride in that and feel that we can learn more. I will address this a bit later, about how to take care of such patients.

A third question is: What do families need now? I think, number one, they need proper diagnostic assessment. That has been touched on several times this morning. We are not perfect, and even it seems that the best of us are wrong about 20 percent of the time. So obviously autopsy is crucial. But we are right 80 percent of the time, and in point of fact, in medicine, that is pretty good. There are many other conditions for which the rate is not even 80 percent in a living person. I think we do have to make a proper assessment of facilities available. Again, as you have already heard, this is also important because 10 or 15 percent of people who have symptoms of forgetfulness in fact have treatable diseases. So we would not want to overlook that. In addition, many patients have medical conditions, like diabetes or heart failure, and if they can be treated, the person at least is more functional.

I think proper diagnosis is very important, not just to make a diagnosis but also because a proper diagnosis also identifies problems. I will return to that in a second.

I think families do need information, some of the kinds of information that Dr. Heston was mentioning was genetics. As you said, many people are quite frightened that maybe this runs in families. To be able to reassure the families in which that does not seem to be true is important. To be able to give the families some idea of what the future might hold I think also gives them something to plan on. Again, it is not a cure, but I think that is important.

A third thing that families need in this realm is help solving problems. We cannot treat the disease in the sense of making it go away, but we can treat specific symptoms. We can treat sleep problems, or we can treat incontinence and make a big difference, or irritability. It seems to simple, but one of the things that clinicians do is teach people how to solve problems. Often, people will come to the doctor, overwhelmed by the whole situation. When you sit down and list what problems they are having, there may be solutions to those specific problems that someone has not thought of. We provide that kind of information. They often get that from other people in the same situation. So we can often make a difference with problem solving, even though we cannot cure the disease.

Another area is emotional support. I think this is a quite crucial element that is being provided now in some part. Another element that I think families need now is some kind of partial relief from care giving. I think that can take various forms. There might be a neighbor coming in for a few hours, or just paying someone. It does not have to be a professional. Paying someone to come in a few times a week. I think day care centers are a tremendous resource for such families, and perhaps we may even need to have units where a person could be hospitalized 1 or 2 weeks a year to give a family a break from care. I think these are quite important.

Finally, there is the issue of financial relief. I do not think there are any solutions to this, but I will make some specific suggestions.

The fourth area that I would like to address is, Is there research needed in this area? We have heard a lot this morning about research into the basic biology and cause and, hopefully, ultimately treatment. I think that must be the major focus of our research effort. However, it is my feeling that if that is the only focus of our research effort, then we are neglecting the needs of many people who are ill right now.

I would like to see, as part of the research that is funded, some focus on how to best care for patients. We are really pretty ignorant. Although I think I might do a good job with something, I have no way of knowing what the best ways are to take care of patients. I think research would clarify that a little bit for us. I think one way to go with that is to look at specific problems. Again, we hear a lot about memory problems in Alzheimer's disease, but when you talk to the families, that is usually not the biggest problem. The biggest problem is the incontinence, the wandering, the sleep problems. Again, it is different from patient to patient.

I believe we need to research how to specifically treat these problems, as well as find a cure. We might be able to help a person sleep all night. That can have a tremendous effect right now, because we do not know when the disease will be cured.

I think we also should be funding some small pilot studies to try to determine whether providing these relief services actually do make a difference. I believe that things like day care and having people come into the house probably can, for many families, delay nursing home placement for 6 months or a year. But that is just a belief. I do think that because health care costs are so overwhelming, if we could demonstrate that we could delay nursing home placement by a year, the cost savings there could be tremendous. So I do think that we should see some funding of research into these things.

Finally, I think we should look into some kind of relief or reimbursement for home care. I do not have specific suggestions about what form that might take, whether it is tax credits or insurance coverage or whatever. But again, many of these are people who have retired. They are on very limited budgets, and even to pay somebody $4 or $5 an hour to come in and sit just one or two afternoons a week, that can be a tremendous relief for this person who is basically providing 24-hour care.

Perhaps if we could in some way provide limited financial relief for people, say in the form of tax relief, to provide that kind of respite relief service. That might have a great positive effect on the families. I would like to urge that research continue into how to take care of the people who have the disease now, while the major research effort continues on finding the cause and, hopefully, ultimately prevention.

Thank you.

[The prepared statement of Dr. Rabins follows:]

ALZHEIMER'S DISEASE AND THE FAMILY

Testimony submitted to Subcommittee on Investigations and Oversight, Committee on Science and Technology, September 20, 1984.

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Mr. Chairman, ladies and gentlemen:

As we have heard this morning, Alzheimer's disease and related disorders are a group of progressive debilitating conditions caused by the deterioration of the brain. The effects of this group of illnesses go far beyond the individual sufferer to reach family, friends and ultimately the community at large. I will try to sketch out how these diseases affect the family by addressing 4 questions.

1. WHAT ASPECTS OF ALZHEIMER'S DISEASE AFFECT FAMILIES?

A. The symptoms of the disease.

Most people have known someone with senile dementia of the Alzheimer's type. What usually has stuck in their minds is that memory loss is severe. In fact, memory loss is often one of the early signs of Alzhemier's disease and one of the very most devastating. However, the illness also goes on to effect many other functions. People with this disease may lose the ability to carry out everyday activities such as using a knife and fork, brushing teeth, or even dressing. This is not just weakness but rather a loss of the brain's ability to coordinate the complex series of movements that go into such acts. Also, patients with Alzheimer's disease often have difficulty communicating their thoughts and understanding what is said to them. This can be a major source of frustration to both the ill person and those caring for him. In the late stages of the disease the patient can become unable to walk and may no longer control the basic bodily functions of waste elimination.

In addition, there are sometimes behavioral symptoms which cause difficulty. Aimless wandering and poor sleep can be seen. Sometimes there are changes in personality and irritability.

As you can imagine, dealing with these symptoms in a loved one can be quite difficult. Families who commit themselves to caring for an Alzheimer victim must not only deal with these symptoms day after day but also with the knowledge that the symptoms will probably be progressive and worsen over time.

Thus the care of the Alzheimer victim requires that families meet the ever changing physical symptoms of the illness. This can lead to fatigue and exhaustion in the caregiver.

B. The emotional effects of caregiving.

In addition to the physical effects of the disease, families must also face the emotional effects of seeing a loved one deteriorate before their eyes. Relationships between people which have developed, grown and evolved over a lifetime are often unalterably changed. It is not surprising, therefore, that symptoms of depression, anger and guilt are commonly reported by family caregivers of Alzheimer's patients.

The care of a patient with this illness can cause major changes in the family's social life. Caregivers often find that it is harder to get together with friends, particularly when two couples had relationships. In addition, the fatigue of caregiving can leave one too tired to enjoy hobbies or outside activities.

C. Financial impact.

Families must also face the financial burdens that Alzheimer's disease brings to bear. Individuals sometimes retire early to care for a loved one. Expenses such as paying someone to stay with the patient while shopping, doing errands or visiting others can add up.

For many families the most frightening financial prospect of all is that of nursing home placement. It is my belief that nursing homes are a necessity for many patients given the severity of their illness but the average cost of a nursing home bed, now $20,000 per year in Baltimore, can be financially devastating. This is only for the bed and does not include doctor fees or other medications that might be needed.

2. WHAT ARE FAMILIES CURRENTLY DOING TO CARE FOR AFFECTED FAMILY MEMBERS?

It is surprising to most people that the majority of individuals with Alzheimer's disease are being taken care of at home. The best estimate is that one-third of the 1 1/2 - 2 million individuals with dementia reside in institutions--the other two-thirds are being cared for in non-institutional settings, mostly at home by families. When we look at who are actually in institutions the data is even more enlightening. Nursing home patients, when compared to impaired people living at home, are more ill, more likely never to have married and more likely to have had no children. There are thus fewer available family to care for them.

Therefore, I believe that we must focus on two "institutions" which are providing care for the Alzheimer victim; the nursing home and the family/community.

In discussions of Alzheimer's disease we tend to get discouraged and overwhelmed by the enormity of problems and lack of absolute solutions. I believe the fact that many people say they want to care for their loved ones and the fact that the majority of Alzheimer victims are actually cared for by family negates the myths that the elderly and ill are abandoned by their families and that the family in this country is breaking down. It also provides a way for us to make a postive difference in the care of Alzheimer disease victim. Teaching families to solve the problems of day to day care and helping a family member to better cope with a difficult situation is a positive contribution which we can make now. The knowledge that this leads to better care is certainly one of the positive aspects of what is currently being provided to Alzheimer victims and their families.

3. WHAT DO FAMILIES NEED?

A. Proper diagnostic assessment.

This is important for several reasons. Pershaps 10-15% of patients who present to doctors with symptoms of forgetfulness are found to have a treatable medical or psychiatric cause of the impairment. They will get better if identified early and treated properly. Further, some patients with dementia suffer from other medical or psychiatric diseases which did not cause the dementia but which may be making the symptoms worse.

Treatment of the coexisting illnesses can also lead to improved thinking and behavior.

A proper evaluation can also help the family identify problems and remaining strengths of the ill person. These can lead to interventions which might improve function.

B. Information.

The evaluation process can be helpful by giving families as definite a diagnosis as possible. This can give them some idea of the future course of the illness and help the family plan for the future. Informed consumers should be able to better use existing resources and pressure for needed changes in care delivery.

C. Help in problem solving.

One of the most important aspects of the assessment is to use the information gathered to solve problems. Such problems as incontinence, feeding difficulties, irritability and poor sleep can sometimes be solved even though the disease itself is untreatable. Teaching the concepts of problem-solving to paid caregivers and families is an important part of what we do professionally.

D. Emotional support.

As discussed above families can become emotionally and physically drained by caregiving. Realizing that they are not alone and that other people have solved some of the problems they face is an important insight for many families. Support groups such as set up by the Alzheimer's Disease and Related Disorders Association and many other agencies can be helpful. A minority of people need and benefit from some more formal counseling. We have also written a book, The 36-hour Day, which attempts to provide some emotional support for families.

E. Partial relief from caregiving.

I believe that partial relief from caregiving is important for the physical and psychological health of the caregiver. Although there is no data to support the contention, I also believe this allows some individuals to stay at home longer than they would otherwise. Relief services can be provided by family, friend and neighbors; a paid non-professional who spends several hours or several days a week with the ill person; day care centers; brief hospitalization units where the patient can stay for one or 2 weeks and allow the family to take one or two weeks a year away from caregiving. I believe we should develop such partial respite services in as rational a manner as possible.

F. Financial relief.

At present there are almost no sources of financial relief for those providing care for a loved one with Alzheimer's disease. Medicare considers the dementing illness to be chronic, denying any support for the expenses of long-term care. Office visit reimbursement is also limited by the ceiling on yearly psychiatric visits.

4. IS RESEARCH NEEDED IN THIS AREA?

While I am committed to caring for individuals who have the dementing illnesses and to helping their families and caregivers as much as possible, I also recognize that there is very little scientific data which to base this practice. While supporting a strong research effort into finding the causes, treatments and prevention of Alzheimer's disease I also strongly believe that we must study how to best care for the patients. We must determine whether we can relieve caregivers and society of some of the negative effects of the disese.

I believe we must continue to support research which looks at specific problems that are caused by dementia. Incontinence, sleep disorder, irritability, and explosiveness, and wandering should be carefully studied and management/treatment methods examined to determine how to best solve these problems.

Pilot studies should be performed to determine whether providing relief services for families delays or speeds up institutional placement of patients.

Careful study should also be given to determine the effects of reimbursement for home care. These could be as tax credits or insurance coverage. In these times of frighteningly escalating medical costs we must carefully study the potential effects of changes in reimbursement before pushing for them. From a clinicians point of view, I believe that some financial support of relief services will have a major positive effect on the families who are already providing a great deal of care to the Alzheimer's disease victim.

Mr. **Gore**. Thank you. We will hold off on our questions, until the panel is finished.

Dr. Carl Eisdorfer from Montefiore Medical Center in the Bronx, we will hear from you next. Thank you for being here.

STATEMENT OF CARL EISDORFER, M.D., PRESIDENT, MONTEFIORE MEDICAL CENTER, BRONX, NY

Dr. **Eisdorfer**. Mr. Chairman and Mr. Winn, I am Carl Eisdorfer, president of the Montefiore Medical Center in the Bronx. Montefiore is one of the Nation's largest voluntary nonprofit medical centers and the primary clinical affiliate of the Albert Einstein College of Medicine. I am also professor in the Department of Neurosciences and of Psychiatry at the College of Medicine. On a personal note, I was the initial cochairman of the Medical and Scientific Advisory Board of the Alzheimer's Disease and Related Disorders Association, and currently a member of its board of directors. They are meeting in Chicago today, and on their behalf I offer you our congratulations and thanks for a job well done and for your interest.

The aging of our society has become one of the great bittersweet successes of this century. People are living longer, which is a tribute to a variety of activities. On the other hand, it has created some problems. Two of the most serious of these are caring for the increasing number of middle-aged and older persons with Alzheimer's disease. You saw the curve. There is almost a doubling of the probability of getting Alzheimer's disease with every 5 years of life past age 65. It is reflected in the curve Dr. Heston gave. The second one is dealing with the family and its role in the caring process.

Until recently, for millions of victims suffering from Alzheimer's disease and their families, they struggled in what one of my patient's husband called "a life of quiet desperation." As recently at 1976, in a movie we did for professional audiences, one daughter said, "Everywhere we went, no one could tell us anything about it. Everything we learned about Alzheimer's disease, we learned from living with it." Now, that has considerably changed. The book, The 36-Hour Day, the media, a variety of other factors have caused change. However, we ought not to be confused. We still know virtually nothing about the life course of Alzheimer's disease itself or of its related disorders. I will talk about this in a minute. We do not even know if Alzheimer's disease is "a" disease or is really a blanket term for a number of diseases. I want to get back to that too, because it may confound some of the basic research.

To explain, Down's syndrome has really two genetic substrates. The concept of pneumonia we understand is fluid in the lungs. Pneumonia can be caused by a range of things, from the pneumococcus to something you swallow to a range of other activities. We talk about cancer, but we know there are many kinds of cancers and so on. We have not yet clearly demonstrated that Alzheimer's disease is or is not a single entity' with a single cause, and we certainly do not know the life course.

That certainly makes caring for the patient, from the physician's point of view or from anybody else's point of view, very complicated. It also makes the management of the patient by the family even more complicated.

The latest evidence supports the idea that many of us recognize, namely that the patient's family, where available, is the foundation of care. Families are the most significant unpaid health and social resource in the United States for Alzheimer's patients. I believe that the family simply is not getting the help it needs. They need information as to the nature of illness, they need information about genetic concerns, they need information about resources in the community, the existence of family health groups, legal and financial advice—and no one has touched on that, and that turns out to be a very key role in the management issue—they need to understand ongoing care and physical changes in the home which could help manage the patient, just to name a few other things. I would certainly support everything that Dr. Rabins said.

Often against the advice of physicians and friends, and with relatively little help from the community, families fight courageously to keep patients at home. They discover by living with it that Alzheimer's patients live for many years after the diagnosis. It is surprising that even today, public policy based in State and national governments seem to believe that families are trying to dump patients in nursing homes, and we build financing accordingly. That is just sheer nonsense.

If anything, those of us providing clinical care see the opposite. Most families care for their own and go to extraordinary lengths to provide care. Indeed, for most individuals, the need to institutionalize a spouse or a parent evokes considerable upset and guilt and, as a rule, reflects an act of desperation.

Families are doing things that defy the textbooks. Many incorporate the patient in a range of activities, and some we have worked with have invented creative household tasks so that the individual patient will feel a part of the ongoing family—the sense of self-worth, the raison d'etre that I think is important for many of us. Caring for a patient with dementia can be an exhausting and overwhelming experience, but many family care-givers do cope for long periods and for some I think you have to say that it becomes a period of significant personal growth, although it may not feel it at the time.

The number of relatives involved in the care of Alzheimer's disease is probably well into the millions. In a recent survey of our own population in New York, the average number of family members living near the patient and directly involved in their care was more than five per target patient. Actually, in one substudy, it was as many as a little over seven individuals. It is thus remarkable that so little time or effort has been devoted in research to try to understand the impact of this tragic disease on the family.

Much of the information we now have on family care-giving is based on data carried out in Great Britain in the sixties and seventies, and involved other diseases—schizophrenia, mental illnesses, and the rest. That research, however, as well as recent studies in the United States, has consistently reported that primary care-givers—the primary care-giver is the one everyone in the family agrees is the key person—shows increased vulnerability to physical illness, emotional disorders, and family conflict. The burden usually falls on the spouse or one of the patient's daughters, typically the oldest daughter, if such a person exists.

The most distressing problems reported by the care-giver are, in order, emotional disturbances of the patient, disruptive sleep patterns, and incontinence of bladder and bowel. We clearly need clinical research in all three areas from a management point of view. It is interesting that Dr. Rabins and I were not in collusion; we did not know of our testimony, and I think Peter said the same thing.

Let me review these quickly. Since dementias are specifically diseases of behavior, with associated cognitive and mental problems, such patients represent among the most difficult human beings for whom to provide care. Patients with Alzheimer's disease do not change their physical appearance, yet there is a real loss of self and an unending series of aggravating, disruptive behaviors which make it difficult for anyone to provide care. One daughter of a patient said to me, "The death of the mind is the worst death imaginable."

Caring for the Alzheimer's patient is caring for an individual who is restless, may become irritable, may have aggressive outbursts, become downright fractious, is an individual who may be depressed, agitated, angry, or simply chronically frustrated and frustrating. Patients wander at all hours of the day or night. The care-giver has a constant fear that the patient will get lost unless they are sequestered or monitored at all times. That is why "The 36-Hour Day" is such an apt phrase.

While the nature of the disease is the inability to remember, it carries with it profound problems of disorientation, agitation, and personal distress. To best explain this is to ask you how you would feel if you woke up every morning not knowing who you are, where you are, or who the people around you were. That is the kind of thing we talk about with families to help them get an understanding of what they are dealing with.

It is one thing to be a parent to a child. It is quite another thing to be a parent to one's own parent, particularly when that is not a voluntary exchange. Many of us have changed diapers, been involved in bowel and bladder training for our kids and have found them not such terrible experiences. On the other hand, it is very different when you have to care for bladder and bowel problems of an older relative, a mother or a spouse who is unable to control themselves and who is wandering around the household. At the same time, these same patients may aggressively resist anything like diapers or control devices.

I do not want to belabor the point, except to remind the committee that Alzheimer's disease is not a disease of weeks or months or even 1 or 2 years. It is a disease of many long years. Once again here, we have behavioral clinical strategies which could work, which we could borrow from other areas, which have not been researched and applied to these patients.

In a study completed in our laboratories and recently reported to the gerontological society, we discovered that of the primary caregivers living at home with a patient, more than half showed signficant depressive symptomatology after approximately a year-and-a-half. That general finding has been confirmed in other places. This is also complicated by the fact that most of our Alzheimer's families report a reduction in social network. In English, this means that friends and other relatives tend to stay away, partly because it is difficult to be in the same household with an Alzheimer's patient, partly because of the embarrassment, partly because it is usually not comfortable for the Alzheimer's patient to be taken for a visit to another place without disruption.

Indeed, one of the ways in which we effectively manage the Alzheimer's patient is by setting up a fairly consistent daily schedule, so that disruptions in their routine are aggravating, and it raises a sort of Catch-22. This gets to be a particular problem around birthdays, holidays, and so on. The Alzheimer's patient really seems to get aggravated by these special events that have so much meaning. One of my most tragic stories is that of a 3-year-old who was having a birthday, and her grandfather walked out just before the cake came in because it was time for him to take his walk.

The data, however, again are clear. Social supports, including friendly visitors, personal support service from the outside, and a variety of interventions can make a major difference in the life of the caretaker and the patient. Remember, too, that in the case of the older Alzheimer's patient, the spouse is also going to be older. This is particularly challenging then because the spouse is likely to have an illness. Certainly this is the case where the spouse themselves is over 60 or 65 years old on a probability basis.

When the patient is cared for at the home of a child, particularly a two or three generational household, there is a high likelihood of marital and family discord. Here, the middle-aged woman, often the patient's daughter, is literally the woman in the middle, torn by conflicting loyalties and responsibilities in both directions.

Until this point, I have been talking primarily about short-term emotional and physical draining. There are serious economic problems as well, and I will touch upon them later. I would like to suggest, though, that we simply do not know the long-term consequences of this burden of love. We know about stress, and we know about the impact of stress, but only in the most general terms. Certainly, in some instances the family may pull together and do well and even grow as a consequence of caring, but unfortunately the evidence seems to be piling up that with the progression of the dementia and the prolongation of the disease, the burden on the family increases. Without resources in the community or that family support, without other help, significant psychiatric and physical health problems occur. Indeed, it is these problems and the associated financial difficulties which lead eventually to a crisis and the need to institutionalize the older relative.

Institutionalization, in our experience, is usually the consequence of some crisis in which the patient takes a modest turn for the worse. More likely, the family finds themselves at the end of their rope, either physically, psychologically, or financially.

What is startling about this is that there is plenty of evidence to demonstrate that psychological, social supports, and education—along with a variety of home and community services—can help the family, help the patient, prevent institutionalization, and reduce long-term costs of care for the Alzheimer's patient. In a national inventory of services just completed this month, it was reported that health care and rehabilitation services are the most commonly established programs to help these patients, primarily because of the availability of health care reimbursement. A relatively small number of mental health and social network supports are reported to exist nationally, and because of a lack of funding, relief services, such as adult day care and respite care, are simply not widely available, despite their demonstrated efficacy, widespread use in other countries, and the need that caregivers have repeatedly expressed for exactly those services.

The survey also revealed that considerable local philanthropic funding for services to the elderly is in fact being used, that active volunteer programs are already in place, and that most agencies want to expand their services in the directions I have described. Certainly, there is a great desire to provide care in the community nationally, on the part of social agencies and hospitals alike. That is the good news. The bad news is that every time we focus on the community, if we do not deliver the supports necessary, it throws an increased burden on the family. What we do bureaucratically is to cut off the other institutional supports.

The best example—and I will not go into details—is what we did in a best possible way to reduce State hospital use for psychotic patients in the sixties and seventies. What we said is we did not want to hospitalize psychiatric patients. We set up a pattern of community-based supports. We denied access to State hospitals to psychiatric patients and then never paid for the community support system. Now we have come up with a horrible tragedy. There are tens of thousands of mentally ill patients who have no home at all. We do not have a pattern nationally of community-based mental health that is really efficacious, and in fact many of the patients who might have been in State psychiatric hospitals are in fact in nursing homes, sustained by psychotropic drugs which may be inappropriate or used in dosages which are probably excessive.

I will specifically target the Alzheimer's patient, and as a side issue, I will tell you we know precious little about the psychopharmacology of Alzheimer's patients. I would warrant that the vast majority of Alzheimer's patients in institutions are on psychotropic medication.

We have another issue. We have targeted health care funding to the patient, which I think is fine. But if a wife brings her husband with Alzheimer's disease to a physician or clinic, there is reimbursement for seeing the patient. But despite the fact that good care would indicate that we spend much more time with the wife or the husband or the child to keep the patient out of an institution, to help keep the family intact, to prevent that spouse from developing an illness, there is no support for such care. Thus, the spouse may be forced to institutionalize the Alzheimer's patient because of their own needs. The result, of course, is incremental costs on Federal Medicare and State Medicaid costs.

The personal and financial toll of caring is in fact not the only problem. For many families, the ability to sustain the patient at home is an important social and moral commitment. By giving no help to families but instead giving a subtle incentive to institutionalize, we go exactly counter to our rhetoric and our belief—and this has been the belief of every administration since I have been aware that there was such a thing as an administration—regarding the importance of the family over the institution. What can be done?

I believe that Medicare and Medicaid as they now exist, however costly, are not adequate to meet the needs of our aged population and their families. It is not the amount of money but the design of the financial programs which are at issue. Medicare was designed to pay for the cost of acute care in hospital settings when it was done in the early sixties. It avoided community and chronic care, however, that typifies exactly what the needs of the Alzheimer's patient and family are.

Medicaid was intended primarily to carry the burden of paying for acute medical services for the young or poor. It was never intended by its founders to be the primary source of funding for skilled nursing home and for other kinds of nursing home care in the United States, and that has now become, as you know, a national problem. Moreover, the virtual absence of private insurance for long-term care in the United States and of funding for any combination of social and health services complicates the problem. It is simply the case that we now pauperize families in long-term care institutional settings and then are forced to provide Medicaid funding when less expensive community-based alternatives could be made available.

Initiatives are needed to examine the possible role of catastrophic insurance for Alzheimer's disease and family support. Alternative forms of group coverage need to be explored—and this may have to be done through special legislation, to allow tax credits for this type of activity.

I think we need to explore support of spouses through tax relief, for caring patients at home as an alternative to institutionalization. We need much more home-based alternatives to long-term care.

Let me identify the Lombardi program in New York, which has played a very valuable role. We need social and personal support, as a component of health-care insurance, and a variety of other devices. Eventually, what we really need is a plan to organize community and medical services for families. We need a bridging of social and preventive health services, and we need to address fundamentally the role of the family in caring for the patient. Thank you.

[The prepared statement of Dr. Eisdorfer follows:]

Testimony to the United States House of Representatives Committee on Science and Technology, Subcommittee on Investigations and Oversight Hearings on Research and Causes, Diagnosis and Cures of Alzheimers Disease

SEPTEMBER 20, 1984

Chairman Gore and distinguished members of the Committee. I am Dr. Carl Eisdorfer, President of the Montefiore Medical Center in the Bronx, New York. Montefiore is one of the nation's largest voluntary, non-profit medical centers and is the primary clinical affiliation of the Albert Einstein College of Medicine. I am also Professor in the Departments of Psychiatry and of Neurosciences at the Albert Einstein College of Medicine. I am a past president of the Gerontological Society of America and of the Western Gerontological Society and was the founding Director of the University of Washington's Institute on Aging as well as formerly Director of the Duke University Center for the Study of Aging and Human Development. For more than twenty-five years, I have been working in the field of gerontology and geriatrics and since 1969 have focussed heavily on research and clinical practice with Alzheimer patients and their families. I was the initial co-chairman of the Medical and Scientific Advisory Committee of the Alzheimers Disease and

Related Disorders Association (ADRDA) and am currently a member of its Board of Directors.

The aging of our society is perhaps the great bittersweet success of the century. People are living longer, a tribute to science, medicine, better economic conditions, improved sanitation and public health measures. However, we face many challenges in dealing with some of the problems imposed by this aging population. Two of the most serious of these challenges are caring for the increasing numbers of middle-aged and older persons with Alzheimers disease and related disorders and in dealing with the family and its role in the caring process.

Until recently, the several million victims suffering from Alzheimers disease and their families have struggled alone living what one patient's husband called "lives of quiet desperation" trying to cope with the impact of the disease. There was a time when families had nowhere to go for help and little if any information to read. In a film we produced in 1975 for professional audiences, one daughter of an Alzheimer victim declared, "Everywhere we went no one could tell us anything about it. Everything we learned about Alzheimers disease we learned from living with it!"

The situation is improving. There are now research and clinical centers developing throughout the country. A number of books, pamphlets, and family handbooks have been and are being written, and features have appeared in newspapers, magazines, television and the radio. People now are aware of the disorder. However, Alzheimers disease remains a challenge for caring. Despite this increased public awareness and the growth of research activity, little has been done to change the course of Alzheimers disease or the other progressive dementias. Yet, a great deal must and can be done for the patient and family to maintain the quality of life at the highest possible level. Caring for the patient by working with the family, an approach which we embarked upon in the 60's is still the best strategy available. If anything, the latest evidence supports the idea that the patient's family, where available, is the foundation of care for the patient with dementia. Families are the most significant unpaid health and social resource in the United States for Alzheimer patients. Not only are families not paid, but oftentimes they must bear a special burden.

The family does not get the help it needs. They need information as to the nature of the illness, their genetic concerns, resources in the community, the existence of family self-help groups, legal and financial advice, and ongoing care. Often instead they are given confusing information about life expectancy and told that institutional care is needed imminently. Often against the advice of physicians and with little help from the community, families fight courageously to keep the patient at home. They discover, by living it, that Alzheimer patients can live for many years after the diagnosis of the disease. Surprisingly, even today sectors of state and national government express the belief that families dump patients into nursing homes. If anything, those of us providing clinical care see exactly the opposite. Most families care for their own and go to extraordinary lengths to provide for their cognitively impaired relative. Indeed for most, the need to institutionalize a spouse or a parent evokes considerable upset and guilt and as a rule is an act of desperation.

Families are doing things that defy the old textbooks. Many incorporate the patient into a full schedule of projects and activities, and some have invented creative household regimes designed to support the individual even as the impairment progresses. They persevere despite increasing isolation from other relatives and friends and continued rejection from many segments of the professional community. Although caring for a patient with dementia can be an exhausting and overwhelming experience, many family caregivers do cope successfully for long periods of time, and for some it becomes an experience that leads eventually to enormous personal growth.

The number of relatives involved in the care of the victims of Alzheimers disease and related disorders is probably in the millions of Americans today. In a recent survey of our own patient population at Montefiore, the average number of family members living near the patient and directly involved in their care was more than five per patient.

It is thus remarkable that so little time or effort has been devoted to try to understand the impact of this tragic disease on the caring family. Much of the information we now have on family caregiving was carried out in Great Britain in the sixties and seventies. That research as well as recent studies in the United States have consistently reported that primary caregivers show increased vulnerability to physical illness, emotional disorders and family conflicts. The burden usually seems to fall on the spouse or one of the patient's daughters, if they exist. It is not one problem but the multiplicity of problems that overwhelm caregivers. The most distressing are those involving 1) emotional disturbances, 2) disruptive sleep patterns, and 3) incontinence of bladder and bowel.

Since the dementias are specifically diseases of behavior with associated cognitive and mental problems, such patients represent among the most difficult persons for whom to provide care. A colleague of mine, Dr. Donna Cohen, and I have recently completed a book entitled The Loss of Self which describes the way patients feel about themselves as well as the way caregivers feel about their relatives. Patients with Alzheimers disease often do not change in physical appearance for years, yet there is a real loss of self and an unending series of aggravating, disruptive behaviors which make it very difficult for anyone providing care.

Both the victim and the family suffer painfully with the inexorable dissolution of self. Loss of sight, hearing, an arm or leg challenge a person to cope with a significant change in self. However, the victim of Alzheimers disease must eventually come to terms with a far more frightening prospect--the complete loss of self. And for the family, according to one daughter, "the death of the mind is the worst death imaginable." Suffering from an incurable illness is something nobody can do for another, but family members endure a life of emotional turmoil and tragedy as they witness the disintegration of thought and language in someone they love.

The suffering is real, but a diagnosis of dementia does not mean a life of exile. A great deal can be done to care for individuals with Alzheimers disease and related disorders throughout the illness, and everyone who is willing can take an active role in their treatment. Resources and therapeutic strategies must be implemented to meet some of the needs of the family and help them cope with the changes in their life. Without assistance the family's chronicle of care often carries them unnecessarily to the limits of physical and mental exhaustion. In a medical education film we produced in 1976 one of the patients declared emotionally, "It's a thirty-six hour day."

Caring for Alzheimer patients is a task as complicated as any we face. Alzheimer patients are restless, may become irritable, and frequently have outbursts when they become downright fractious. They may also be depressed, agitated, angry, or simply frustrated and frustrating. Patients may wander at all hours of the day and night. Constant fears that the patient will get lost unless they are sequestered or monitored all the time is wearing--particularly over the many years of the disease. The nature of the disease is the inability to remember, and it consequently carries with it profound problems of disorientation, agitation and personal distress. As we tried to explain to families in an effort to help them understand the disease: How would you like to wake up every morning not knowing who you are, where you are, or the identity of those around you? Dementia takes its toll on the patient and anyone attempting to care for the patient.

It is one thing to be a parent to a child. It is quite another thing to be a parent to one's parent. Many of us have changed diapers, been involved in bowel and bladder training, and have found them not such terrible experiences. It is, however, much different when you have to care for the bowel and bladder habits of older adults who are unable to control themselves and who may wander about the home. At the same time some patients aggressively resist diapers or other control devices. I don't think we need to belabor the point, except to remind the Committee that Alzheimers disease is not a disease of weeks or months. It is a disease of years--many long years.

In a study completed at Montefiore and recently reported to the Gerontologic Society we discovered that of the primary caregivers living at home with the patient more than half showed significant depressive symptomatology. That general finding has been confirmed at other places. It has also been repeatedly reported, though not yet confirmed in the literature, that the Alzheimer family has a reduction in their social network. This would mean that friends and other relatives tend to stay away partly because it is difficult to be in the same household with Alzheimer patients, partly because of embarrassment, and partly because it is usually not comfortable for patients to visit new places without disruption. Indeed, one of the ways in which Alzheimer patients are effectively managed is by setting up a fairly consistent daily schedule. Disruptions in their routine do seem to be aggravating. The upshot of all of this is that the heaviest burden of care falls directly on the family and at a time when it loses increasingly the ability to sustain itself. The data, however, are clear. Social supports, including visitors, and personal supports can make a major difference.

The husband or wife, where available, is usually the primary caregiver for the older person with Alzheimers disease or related disorders. This can be particularly challenging if that spouse has their own illness, and in most instances there is some chronic illness already existing in adults over sixty-five. When the patient is cared for at the home of a child, there is the prospect of marital discord within that home, and particularly in three-generational households, marital and family conflict is reported as a consequence of care. Here the middle-aged woman, often the patient's daughter, is literally the woman in the middle torn by conflicting loyalties and responsibilities.

Until this point I have been talking primarily about the short-term emotional and physical drain of caring. It must be understood that, in fact, we have really only scratched the surface. We simply do not know about the long-term consequences of this burden of love. Certainly, in some instances the family pulls together, does well, and there is even emotional growth as a consequence of caring. But the evidence seems to be piling up. As the dementia progresses and the patient deteriorates, the burden on the family increases, and without resources in the community and other family supports, family members may develop significant psychiatric and physical health problems. Indeed, it is these problems and associated financial difficulties which lead eventually to a crisis in caring and the need to institutionalize an older relative. Institutionalization is usually a consequence of some crisis in which the patient takes a modest turn for the worse, or, more likely, families find themselves at the end of their rope either physically, psychologically or financially.

What is startling about all of this is that there is more than enough evidence to demonstrate that psychological and social supports along with a variety of home and community-based services can: a) help the family, b) help the patient, c) prevent institutionalization, and d) in all likelihood, reduce the long-term cost of care for the Alzheimer patient. In a national inventory of available services to help the family care for older persons, Martha K.

Parker of Interage reported earlier this month that health care and rehabilitation services are the most commonly established programs, primarily because of the availability of health care reimbursements. A relatively small number of mental health and social support services are reported to exist. Relief services such as adult day care and respite care are not widely available despite their demonstrated efficacy, wide-spread use in other counties, and the demand that caregivers have expressed for those services. The survey also revealed that considerable local philanthropic funding for services to the elderly is available, active volunteer programs are in place, and most agencies want to expand their services not only to deal with acute care problems but also to try to provide adult day care, respite and social services. When we publicized the current program of the Robert Wood Johnson Foundation on Initiatives on Long-Term Care for hospitals throughout the United States, over 400 hospitals in the community in virtually every state indicated their interest in developing noninstitutional long-term care alternatives in the community.

Clearly, there is a great desire nationally to provide care in the community, not only to make it less expensive, but also to significantly improve the quality of life over institutional-based care. That is the good news. The bad news, of course, is that the focus on community alternatives could well put an increasing burden on the family. It is now well recognized that our nation's community mental health programs, which were promoted to reduce state hospitalization of psychiatrically impaired persons, many of whom had Alzheimers disease, have been a frightening failure. This is because we restricted access to state hospital care for many young and aged patients. However, the pattern of Federal and state funding for psychiatric care, particularly for care of the aged, never replaced those state hospital programs with community-based alternatives necessary to maintain these patients with a decent standard of care outside the state hospital institutional setting. Many of these patients are now in nursing homes without the benefit of psychiatric, psychological or social work care. They are sustained by psychotropic drugs which may be inappropriate or used in dosages which are probably excessive, but which reflect on the stress of an otherwise burdened nursing staff.

The oldest part of our population is continuing to age rapidly. It is also projected that with every five years of increasing longevity past sixty-five the probability of acquiring Alzheimers disease or a related disorder doubles. The need then to address this problem and the burden of caring on the family and the community becomes an imperative one.

We have, by the nature of Medicare reimbursement, severely restricted psychiatric support for Alzheimer patients and also provide no help to families caring for the patient. That is to say, if a wife brings her husband with Alzheimers disease to a physician or clinic, there is a fee at the Medicare level for seeing the patient, but, despite the fact that much more time will be spent with the wife to sustain her in keeping the patient out of an institution and to prevent her from developing illness, there is no support for such care. Thus, the spouse who may suffer from a variety of psychological and eventually physical stresses may lose the capacity to sustain a relative and may be forced to institutionalize the Alzheimer patient. The result is that the incremental cost on Federal and state Medicaid costs become much higher.

The personal and financial toll of the caring for the diseased victim is in fact not the only problem. For many families the ability to sustain the Alzheimer patient at home is an important social and moral commitment. By giving no help to families but instead giving greater incentives to institutionalize, we go exactly counter to the rhetoric and belief of every Administration during my lifetime regarding the importance of the family over the institution. What can be done?

It has become apparent that Medicare and Medicaid as they now exist, however costly, are not adequate to meet the needs of our aged population and their families. It is not the amount of money but the design of our financing programs which are at issue. Medicare was designed to pay for the cost of acute care in hospital settings, and it avoided the type of chronic care that typifies the needs of the Alzheimer patient and family.

Medicaid was also intended primarily to carry the burden of paying for acute medical care, but for the poor younger patient. However, because of the way Medicare was structured to only meet the acute care needs of the aged, and the virtual absence of private insurance for long-term care in the United States, Medicaid has become the principal payer for long-term institutional care in the United States. Thus, we pauperize such families in long-term care institutional settings and then provide Medicaid financing when less expensive home-based alternatives could be made available.

Initiatives are needed to examine the possible role of catastrophic insurance for Alzheimers disease. Alternate forms of group coverage may also be possible. Special legislation to allow for the support of spouses through tax relief for caring for patients at home as an alternative to institutionalization should be tried. Since family care is far and away the most cost effective way of helping patients, it should be supported and encouraged. Today families receive virtually no help in their difficult task.

Social support as a component of a health insurance program--or prepaid group plans--should be developed as another approach to helping maintain the Alzheimer patient at home. Since such services can also be preventive in nature and help keep patients out of costly institutions, this is also an investment which could lead to important savings as well as an improved quality of life for the family. The role of respite care, in which families caring for patients can have relief for one or two weeks with needed vacations, hospice care as an alternative to dying in isolated high technology hospital settings, trained geriatric social workers working as a team with doctors and nurses to help provide care at home, day care centers, sheltered housing and similar social and environmental supports have all been demonstrated to successfully keep patients out of nursing homes. Eventually, a plan to organize such community and medical services for families will have to be undertaken. This briding of social services and preventive health care will do much to alleviate the burdens of cost and of caring. It only demands that we take on these initiatives.

The goal should be to avoid change in current policy that places hardships on families with cognitively impaired relatives, to develop policies that provide alternative ways of strengthening families, and to provide the options families need to cope with their self-imposed burden of caring. Caring and the value of human life are a part of our moral fabric. With the presence of increasing numbers of persons with irreversible disease in an older population, already considered by many to be surplus to the productive capacity of the nation, the pressure to rethink our moral contract has already achieved national attention. Certainly it is important to deal with the challenge of helpless individuals and the burden on the family.

In summary, we need to recognize that a new and special effort is required if more and more Americans are not to be pauperized or made ill while trying to care for their loved ones, and sadly, though not surprisingly, it may have to be the families themselves who need to place these issues before the decision makers.

Mr. GORE. Thank you very much. We appreciate it. Again, we will hold our questions.

Our final witness is Mr. Glenn Kirkland with the Alzheimer's Disease and Related Disorders Association, Baltimore/Central Maryland Chapter. We are delighted to have you here. You represent a great many people, insofar as this hearing is concerned. I have learned over the course of this investigation how important the association has been to families who have not been able to find help and understanding and companionship and kindred spirits anywhere else. We are just delighted to have you here and to have your testimony. Please proceed.

STATEMENT OF GLENN KIRKLAND, PRESIDENT, BALTIMORE/CENTRAL MARYLAND CHAPTER, ALZHEIMER'S DISEASE & RELATED DISORDERS ASSOCIATION

Mr. KIRKLAND. Thank you, Mr. Chairman. I appreciate the opportunity to speak to this important body. In addition to being the president of the Baltimore chapter, you might be interested to know that I am on the Governor's task force. I have also played a minor role in the TV program that you may have seen, "Living With Grace." That will give you some of the background.

Seven years ago, my wife, Grace was diagnosed as having Alzheimer's disease, and I went through the emotional experiences which I am sure you have heard from other witnesses: fear, anxiety, and general concern. I have seen this beautiful woman deteriorate—I am sorry.

Mr. GORE. That is perfectly all right. We fully understand. You just take your time. We had a hearing in Johnson City, TN, earlier, and we had some very emotional testimony there. In fact, one of our witnesses from Johnson City is here with us at this hearing. We have learned that it is an emotional subject, so you just proceed at your own pace.

Mr. KIRKLAND. Fortunately for me and for many others across the country, help and emotional support and social support are now becoming available.

I want to tell you that late in 1979, Dr. Robert Butler, who was then at the National Institutes of Health, invited a number of individuals to meet in Bethesda, MD, to consider the formation of an organization which would speak with one voice for the victims of Alzheimer's disease. Among the group were physicians, research scientists, and other professionals, and representatives from seven different community organizations. Out of that meeting came the Alzheimer's Disease and Related Disorders Association that you now know. It soon drew support and got the interest of hundreds of communities throughout the Nation. What began less than 5 years ago has now grown to a national organization of more than 100 chapters, with more than 360 family-support groups. This represents a remarkable growth, in my mind.

Two of the most important chapters are in the home State of Tennessee, the State of our distinguished chairman.

Looking back on this, I think this is nothing short of a miracle. As Ben Gurion said, "Those people who do not believe in miracles are not realists." The fact that this organization has grown with the speed it has I think is really remarkable.

Alzheimer's disease has, over the past, been cloaked in a shroud of silence, either out of ignorance of embarrassment on the part of those who had a victim in the family. For whatever reasons, the family would not want to talk about the matter publicly. Fortunately, that is changing now that Alzheimer's disease is becoming a household word. Today, through the results of ADRDA, millions of Americans have heard of Alzheimer's disease. Through the education and awareness activities, people are beginning to understand that Alzheimer's disease is not an inevitable part of the aging process. It is not a natural result of growing old. Indeed, it is a widespread and growing problem, as you all have heard.

Here at the Federal level, ADRDA has helped focus more attention on the need for the greatest emphasis on medical research. Through your constituents, you have heard how serious the need is for medicare coverage, for disability benefits, for veterans' care, and for family support services. Despite the competing pressures that Congress must contend with, you have begun to respond to this need, for which we are grateful. This year, Congress seems well on its way to appropriating more than $50 million for research to be sponsored by the National Institutes of Health, and you have approved the establishment of 10 specialized research centers throughout the country. This is a far cry from a few years ago when the total amount spent was less than $4 million.

ADRDA has carried out a strong and deliberate outreach and education program, targeted to the health professionals and organizations concerned with the care of the elderly. As a result, far more physicians and other health care personnel now recognize the serious nature of the disease. ADRDA, under the leadership of its president, Jerome Stone, has launched a research program of its own. Over the past 2 years, the association has awarded more than $450,000 to 38 researchers in the field, and they have a faculty scholarship award program which is for bright young research people who have committed themselves to basic or clinical research on degenerative brain disorders. Finally, ADRDA has sponsored an autopsy network of three brain banks to provide researchers with tissue that is so vital to the needs of their work, as you have heard this morning.

We are mindful and appreciative of the work of the Administration on Aging, what it is doing, particularly in regard to expanding the concept of support groups through the use of agency personnel. There is a need, however, to expand the services to the caregiver, as Dr. Rabins and Dr. Eisendorf said.

Relief from the overburdening task of care is important if we want to lighten the load on the nursing homes. In-home help, day care for short-term custodial care, is becoming a practical need.

Mr. Chairman, we have come a long way, and we certainly have a long way to go. But as you have seen in the recent media reports, scientists are beginning to uncover some of the mysteries surrounding this disease.

But the heart and soul of the ADRDA lies in its family-support groups. The true spirit of self-help concept is embodied here, where there is no shortage of care or understanding, where families can go for support and solace.

Let me say a word about the Baltimore/Central Maryland Chapter. In 1979, the staff at Johns Hopkins saw the wisdom of having caregivers get together to share their problems. Those of us whose patients were part of the Hopkins medical research program were brought together, and we were encouraged to share our problems and the solutions to our problems. Let me turn that around and say our challenges and the solutions to our challenges. Out of that came our local chapter. As soon as the ADRDA coalesced into a national organization, we joined that organization.

Our goals are very much similar to the national organization. We want to educate the public. We want to show concern to the victim and the caregivers. And we would like to further research. We want to make Alzheimer's disease a household word, and I think we are succeeding. I think the news media have expressed interest, and this scourge is getting good coverage in the press. Although there is no cure and no preventive measure, there is not even a palliative, I am personally optimistic that the medical community is awakening to the fact that Alzheimer's disease is really a silent epidemic. There are a number of significant research advances and new research tools are coming into play, as you have heard this morning.

Mr. Chairman, I hope this testimony has shed some light on the kinds of help we need to provide to families coping with this cruel affliction, and I hope we can count on you and the members of this committee for continued support.

For the record, I would like to leave with you a copy of the ADRDA's national plan to combat Alzheimer's disease, and I will be glad to participate in the question period and see if I can answer any questions you might have.

[The prepared statement of Mr. Kirkland follows:]

Statement by Glenn Kirkland
before the
Subcommittee on Investigations and Oversight
September 20, 1984

Mr. Chairman and members of the subcommittee -

My name is Glenn Kirkland. I am President of the Baltimore/ Central Maryland Chapter of the ADRDA, the Alzheimer's Disease and Related Disorders Association.

I would like to begin by thanking you for the opportunity to appear before this distinguished panel. This is a very busy time for Congress, I realize, but I want you to know that we do much appreciate your concern in this area.

Mr. Chairman, seven years ago my wife, Grace, was diagnosed as having Alzheimer's disease. I suppose the feeling and the emotions I experienced are not unlike those which you have already heard expressed today by other witnesses. I, too, have had my share of dark moments, watching helplessly as a beautiful, healthy, intelligent woman gradually turned into a shadow of her former self.

Alzheimer's disease, as you know, manifests itself in many different ways. Perhaps the one common denominator is the fear, the anguish, and the concern of the family. Each and every care giver has a lot of questions -- but not many answers.

Fortunately for me and thousands of other families across the country, help and support is available.

Late in 1979, Dr. Robert Butler of the National Institute on Aging invited a number of individuals to Washington to discuss the need for a national organization to speak with one voice for families and loved ones struggling to cope with the nightmare of Alzheimer's disease. Among the group were physicians, research scientists, as well as representatives from seven community organizations. The Alzheimer's Disease and Related Disorders Association evolved out of that meeting. It soon drew support and sparked interest from hundreds of communities throughout the nation. What began less than five years has since grown into a national organization of more than 100 chapters with more than 360 family support groups. Today, for example, ADRDA has member organizations in each of the states represented by the distinguished members of this subcommittee. In fact, two of our strongest chapters are located in the Chairman's home state of Tennessee.

Looking back, Mr. Chairman, the fact that these family support centers ever got off of the ground is no small miracle. But as David Ben-Gurion once said, "These people who do not believe in miracles are not realists." You see, for decades Alzheimer's disease and related disorders have been cloaked in a shroud of silence out of ignorance or embarrassment. Whatever the reasons, families would not talk about their struggle; in many respects, they suffered in silence much like the victims of this horrible disease do.

Today, largely as a result of ADRDA's efforts, millions of Americans have heard of Alzheimer's disease. What is more, through education and awareness activities, people have begun to understand that Alzheimer's disease is not the inevitable part of the aging process -- it is not the natural result of growing old. Indeed, it is widespread and a growing problem.

Here, at the federal level, ADRDA has helped to focus more attention on the need for greater emphasis on medical research. Through your constituents you have heard just how serious the need is for Medicare coverage, for disability benefits, for veterans' care, and for family support services. Despite the competing pressures Congress must contend with, you have begun to respond.

This year, Congress seems well on its way to appropriating more than $50 million for research to be sponsored by the National Institutes of Health. You have also approved the establishment of 10 specialized research centers throughout the country. This is a far cry from a few years ago, when less than $4 million was allotted for research on Alzheimer's disease.

In addition, ADRDA has carried out a strong and deliberately outreaching and education program targeted on health professionals and organizations concerned with the needs of the elderly. As a result, far more physicians and other health care personnel now recognize the serious nature of this disease.

ADRDA, under the leadership of its president, Jerome Stone, has also launched a research program of its own. Over the past two years, the Association has awarded more than $450,000 to 38 researchers in the field. We also have a Faculty Scholar Award program to support bright young researchers who have committed themselves to basic or clinical research on degenerative brain disorders.

Finally, ADRDA sponsors an Autopsy Network and three Brain Banks to provide researchers with tissue that is so vital to their work.

We are mindful and appreciative of the work that the Administration on Aging is doing, particularly in regard to expanding the concept of support groups through the use of agency personnel. There is a need, however, to expand the services to the care giver. Relief from the overburdening task of care is important if we want to lighten the load on nursing homes. In-home help, day care as well as short-term custodial care, is becoming a practical need.

Mr. Chairman, certainly we have a long way to go, but as you have seen in recent media reports scientists are beginning to uncover some of the mysteries surrounding this disease.

But the heart and soul of ADRDA lies in its family support groups. The true spirit of the self-help concept is embodied here, where there is no shortage of care or understanding -- where families can go for support and solace.

Let me say a word about the Baltimore/Central Maryland Chapter. In 1979, the staff at Johns Hopkins saw the wisdom of having care givers get together to share their problems. Those whose patients were part of the Hopkins medical research program were brought together. Suddenly, each of us realized that we were not alone. Other people were struggling, too. As soon as the ADRDA coalesced, we joined. Our goals, like those of the national organization are to educate the public, to show concern for the victim and care givers, and to further research. We want to make Alzheimer's disease a household word -- and I believe we are succeeding. The news media is now interested and this scourge is now getting good coverage in the press. Although there is as yet no cure, there are no preventative measures, there is not even a palliative, I am personally optimistic that the medical community is awakening to the fact that Alzheimer's disease is really a silent epidemic. There are a number of significant research advances and new research tools are coming into play.

Mr. Chairman, I hope this testimony has shed some light on the kinds of help we provide to families trying to cope with this cruel affliction. I hope we can count on you and the members of this subcommittee to continue your support in this effort.

For the record, I would like to leave you with a copy of ADRDA's national plan to combat Alzheimer's disease.

I would be glad to attempt to answer any questions you may have, Mr. Chairman.

Thank you.

Report for the U.S. House of Representatives
Committee on Science and Technology
Subcommittee on Investigations and Oversight

ALZHEIMER'S DISEASE

The central and peripheral nervous systems are composed of complex assemblies of nerve cells (neurons) which communicate information by electrical and chemical signals. Neurons directly influence other neurons by liberating specific neurotransmitters which excite or inhibit target nerve cells. Specific types of neurons in distinct regions of brain use different neurotransmitters, and each group expresses specific functions. For example, cholinergic neurons in the basal forebrain give rise to projections to the cortex; by liberating acetylcholine, these projections excite cortical target cells. Degenerative neurological diseases, including Alzheimer's disease (AD), Parkinson's disease, and amyotrophic lateral sclerosis, selectively affect specific neuronal populations -- a phenomenon referred to as "selective vulnerability". The most common disease of this type is AD. Elderly demented individuals represent more than 10% of the population over 65 years of age, and approximately 50-60% of these patients suffer from AD. As our aged population triples in the next 75 years, the costs for caring for affected individuals will rise at dramatic rates, posing enormous ethical, economic, and social burdens.

Clinical Manifestations

Although dementia occurring in old age was clearly described in ancient times, the disease was not documented in the medical literature until 1907, when Alois Alzheimer described clinical and pathological findings in a middle-aged woman with a progressive dementia. AD frequently starts with gradual loss of memory, followed by progressive deterioration of thought, judgment, and the ability to manage personal affairs. Many patients have problems with language skills, praxis, and visual-spatial perceptions. Eventually, these individuals become severely demented.

Diagnosis

At present, there are no laboratory studies specific for AD. The diagnosis is one of exclusion, and a definitive diagnosis can only be made at autopsy. In the future, new imaging techniques, which allow examination of regional changes in metabolism and transmitter-specific circuits, should provide new insights into the relationships of clinical symptoms and brain abnormalities.

Research on Brain Pathology

Significant progress has been made in identifying changes in neurotransmitter systems in the brains of individuals with AD and in demonstrating relationships between these neurotransmitter abnormalities and histological hallmarks of the disease, i.e., neurofibrillary tangles, senile plaques, and loss of specific populations of nerve cells.

Alterations in Populations of Nerve Cells

Changes occur in a variety of neurotransmitter circuits, including cholinergic, somatostatinergic, and monoaminergic systems. Neurons within the basal forebrain provide the major cholinergic innervation of cortex and, in AD, dysfunction and/or death of these neurons appear to be responsible for reductions in forebrain cholinergic markers. Somatostatin levels in cortex are reported to be decreased in AD, and some intrinsic cortical neurons degenerating in this disease may use this peptide as a neurotransmitter. A number of patients with AD show changes in monoaminergic systems. Degenerative changes also occur in nerve cells of the amygdala, hippocampal formation, and neocortex, but the transmitter specificity of these affected neurons has not been clarified.

Neurofibrillary Tangles

In AD, cell bodies of certain neurons often contain neurofibrillary tangles, consisting of accumulations of unusual cytoskeletal elements. The presence of abnormal filaments may alter the dynamics of intracellular transport processes and have profound consequences on neuronal function.

Senile Plaques

Located in the cortex, these abnormal structures are made up of enlarged axons, synaptic terminals, and amyloid; plaques represent foci of abnormal interactions between neurons. It is not surprising that, when sites of chemical communication between nerve cells are disrupted, affected individuals show dementia. Until recently, little was known about the transmitter specificity of elements in senile plaques. Recent studies indicate that cholinergic, somatostatinergic, and monoaminergic systems as well as, possibly, other neuronal populations participate in the formation of senile plaques.

Experimental Studies in Animal Models

Animal models are extremely useful for investigating important questions raised by studies in AD. Several examples illustrate this point. Macaques with experimental lesions of the basal forebrain cholinergic system (e.g., one of the abnormalities in AD) show impaired performance on certain memory tasks. Aged monkeys, who have memory deficits similar to those occurring in aged humans, show cortical senile plaques. Recent studies have demonstrated that neuronal elements in plaques are derived, in part, from neurons in the basal forebrain cholinergic system, the brainstem monoaminergic system, and several intrinsic cortical systems, including somatostatinergic neurons. Thus, the use of animal models has greatly helped to clarify the consequences of specific neuronal injury on behavior and the roles of specific transmitters in plaques, similar to those occurring in AD.

Etiology

Genetic/Chromosomal Abnormalities

In some families, AD appears to be inherited as an autosomal dominant disorder. First-degree relatives of an affected proband have an increased chance of developing the disease. Relatives of AD patients have increased incidence of Down's syndrome. Moreover, individuals with Down's syndrome who survive into the fourth decade frequently develop the dementia and brain abnormalities of AD.

Infectious Agents

It has been suggested that AD may be caused by a transmissible agent because of certain resemblences between brain abnormalities occurring in AD and those occurring in Creutzfeldt-Jakob disease (a dementia syndrome in humans) and scrapie (an encephalopathy in sheep), both of which are caused by transmissible agents with unique physical/chemical properties. However, these transmissible agents have not yet been demonstrated in the brains of individuals with AD, and a report of experimental transmission of the disease to primates has been retracted.

Toxins

The roles of external toxic agents in AD are not clear. Original reports of increased aluminum concentrations in the brains of patients affected by AD have not been confirmed. Aluminum is present in increased amounts in the nuclei of some neurons showing neurofibrillary tangles, but it is not known whether this change is a primary abnormality or a secondary phenomenon with aluminum deposited in already damaged neurons.

Therapy

At present, there is no specific treatment for the cognitive impairment in AD, although supportive care and management of secondary symptoms can significantly improve the quality of life for both patient and family. A variety of therapeutic approaches has been tried in attempts to correct the cholinergic deficiency. These include: attempts to increase available acetylcholine at synapses by providing precursors, such as choline and lethicin; efforts to slow down the breakdown of acetylcholine by using cholinesterase inhibitors; and direct stimulation of cholinoceptive neurons with muscarinic agonists, such as arecholine. Although some improvements on specific neuropsychological tests have been demonstrated with these approaches, none of these therapies have been reported to consistently improve skills necessary for daily living and, thus, they do not appear to be highly useful in a clinical setting.

The development of new therapeutic strategies for AD will be enhanced by further knowledge of transmitter populations at risk in individual patients and by understanding the consequences of these neuronal changes on the behavior of the patient. For example, new therapies may be designed to enhance (by drugs or trophic factors) or replace (by neural grafts) functions of remaining neural circuits. A better definition of the nature of degenerative processes (i.e., which neurons die when in which patients) will also likely lead to better understanding of the causes and mechanisms injuring these groups of neurons. With this information, we will be in a better position to deal with the approaching epidemic of AD.

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Mr. Gore. Thank you very much.

One of the major issues facing Alzheimer's victims and families is financing long-term care. I would like to ask you to evaluate the various funding options for each of the following: Private insurance, and home and community-based care.

Dr. Eisdorfer, in the case of private insurance as an option for funding long-term care, would this require Government money in order to make private insurance feasible? How would you evaluate that?

Dr. Eisdorfer. The answer is, it would probably involve some incentives at this point. I have been working on the problem for about 3½ to 4 years. There are a number of technical issues involving how long do you need to insure a group for the relative risks and so on. There has to be some incentive right now. It has to be done with so large a group across so many ages that they can have a base of coverage, or there needs to be some special incentives.

I think the other component that is important in a private insurance scheme would be to elaborate the base. Let me tie it in to the other question you asked, because I think it is a very important concept. Montefiore is the place where hospital home health care was invented, back in the forties. We also run a large home health care program. Last year we made well over 150,000 home care visits. In any given day, we were seeing over 200 patients in long-term home health care.

We were able to provide long-term home health care, maintaining a patient in the community for about one quarter of what it cost to maintain the same person in a nursing home. The measured functional deficits of the patients we were caring for are not different than the patient in the nursing home. I alluded to this Lombardi legislation. I think if the staff has not already looked at it, I humbly suggest that is a program that ought to be evaluated.

If an insurance scheme then is made broadly enough to include those sorts of options—and some insurance scheme do not, which is part of the problem, I think, which is, it forces you into an institutional mode and therefore runs the cost of care up—if options are put, if this is at least given some initial subsidy in the form of tax relief or something like that to the company, I think that we could get insurance moved out modestly rapidly. Clearly, the need is there. Just as we had to do with catastrophic insurance for cancer, I think there had to be an initiative and finally it was seen to be effective.

Mr. Gore. Could Medicaid waivers be used to help fund home and community-based care?

Dr. Eisdorfer. Absolutely.

Mr. Gore. Would you recommend that?

Dr. Eisdorfer. I would recommend it highly. I would also recommend that as part of that, we rethink what care involves. Part of the difficulty is that we tend to hang our payment scheme on diagnoses and on specific elements of what I would call medical or nursing care, rather than looking at this from a functional point of view. The result is that we cut off social and other community services as part of the care, and we wind up using more expensive medical and nursing care when much less expensive care can be provided.

I think that if we recognize the value of a range of services—like respite care, which I think is a superb case in point. Congress was great in hospice care. I do not want to get involved in politics, but then the Administration changed the rules slightly in terms of the payment, but the hospice concept, here was a more effective way to do it, desired by patients, desired by families, desired by doctors, and cheaper. It took a long while to get that through because it was new. I think we have the same problem. I think the social concept of an HMO, for example, the social component of caring—and that was the survey I just reported, that the interstudy group in Minneapolis came up with—that we have enough medical care in place. It is the other components of care that are forcing the patient into the institution. It is rarely the medical problem that forces institutionalization.

I would also say, in a State like New York—I am on Senator Lombardi's task force—about 5 percent of the patients, for example, in New York State hospitals—maybe as high as 7—are in those hospitals because there are not nursing home beds available, and that drives the costs up even further. Now we are paying for those costs.

Mr. GORE. In addition to granting Medicaid waivers to help with home and community-based care, what other recommendations would you have for actions by the Federal Government, in terms of Medicare and Medicaid, to address the long-term care of Alzheimer's disease patients and their families?

Dr. EISDORFER. Clearly, I think we have to change the rules for reimbursement. Let me give you a couple of examples personally. I have only recently come to the east coast from the west coast, where I was in Seattle. I ran into difficulty in Medicare because when patients were coming to me, and I felt they were on too much medication, and I felt that they needed hospitalization to get off the medication, the hospital and I were not being reimbursed. It was not a personal issue; I was on full-time salary anyway. The hospital was not being reimbursed for me to take patients off medication, which I will share with you, Congressman, is the best thing I could have done for the patient, and that is to get rid of their drugs. But I could not have gotten reimbursed because I was not doing anything.

This may sound silly, but the insertion of a Foley catheter in a patient is Medicare-eligible; the removal of that Foley catheter is not. If you know what a Foley catheter is, you probably winced as I said that. It is bad enough to get one put in without talent. It is a hell of a lot more uncomfortable to have somebody take it out.

Mr. GORE. It would appear to be a useful and, therefore, reimbursable procedure for someone who needed one removed.

Dr. EISDORFER. What has happened is that in order to save money, we have had a series of decisions that restricted care to a procedure-oriented care and to a more hospital-based care. As I say, I run one of the largest hospital complexes in the country, so I am always in favor of being adequately supported. But I would like to develop community programs, and I have difficulty providing funding for those community programs because of the current restrictions. I think there are alternatives, and I think a task force that could look at alternative ways of funding these programs on the national scene would, at this point when we are reevaluating the whole Medicare/Medicaid financial crisis, be an important thing to do historically.

Mr. GORE. Let me give the other two witnesses a chance to respond to that question, and then I want to recognize Mr. Winn. I will come back for some other questions.

Dr. Rabins.

Dr. RABINS. I guess I would really agree with what Dr. Eisdorfer said and just reiterate a bit what I had said. I think that if we could recommend supporting research into what kinds of interventions, like day care for example, whether they can really delay nursing home placement and even effect a savings, I think would be quite important. I do not really have any other specific suggestions.

Mr. GORE. Fine. Mr. Kirkland.

Mr. KIRKLAND. I do not think I have anything to add to what these gentlemen have said.

Mr. GORE. The final recommendation that you made, Dr. Eisdorfer, to have a task force study these options, certainly seems to me to be emminently reasonable. I do not want to make a comment that can even be construed as political in the context of these very intense hearings. But of course it is worth noting that today—I believe it is today—we saw the deletion of a recommendation that a task force look at these options as the major study on Alzheimer's options was released by the secretary. I will come back to that, but I do not want to take up too much time.

Mr. Winn.

Mr. WINN. Thank you, Mr. Chairman. I want to clarify that. I was going to point out that Mrs. Heckler is releasing the task force report at the Alzheimer's Disease and Related Disorders Association annual meeting in Chicago this afternoon. We have a couple of copies, and I have only just thumbed through it, based on some of your discussions. But I think you gentlemen will be very pleased with some of the recommendations that are in here, and you might well have had some input into some of the individuals that were on this task force. I think we all look forward to this and the expansion of it.

Dr. Rabins, I, too, want to thank you for the work on "The 36-Hour Day." It is a fantastic book, and in our case, it was very helpful to our family. Believe me, it was. When the chairman mentioned it, I saw several other heads nodding affirmatively out there in the crowd.

As a matter of fact, Mr. Chairman, I want to compliment you for holding these hearings, even though I sort of prodded you along to maybe embellish them and enlarge them a little bit. I think we have served a good purpose. Some of the witnesses have made some strong points that need to be followed through. Thank God for some of the doctors and professionals that are here, that we do have people who understand the disease and have dedicated much of their life and their future to that.

I do not believe the word "group therapy" as such ever was mentioned. I think that obviously that is certainly one of the many ways that a family can benefit. In the Kansas City area, our family went through great group therapy. Your book was handed out at that time, and that is the way that one session sort of developed.

I think when we hear witnesses like we did yesterday and like Mr. Kirkland a little while ago tells some of the experiences that other people, spouses and other families have experienced, I think that probably helps offset the jolt that you get when you are first informed that someone that you love has Alzheimer's. Group therapy is important and, I have noticed here, it is one of the many answers or suggestions that should be studied further and probably improved. My guess is that even though ADRDA has some suggestions and does a lot of this, there is probably not a basically consistent program around the country. It will probably vary from time to time, depending on the amount of training those involved have, and the amount of meetings that they have attended and the ideas that they have picked up and what is possible from a financial standpoint.

It just dawned on me when the chairman and Dr. Eisdorfer were talking about how do we fund this, it just dawned on me that probably a problem—and maybe it has been discussed; I have never heard it discussed before, but after listening to the problems of the financial drain on the families involved—and we had some testimony to that effect yesterday—there are a great many families that would probably like to help and set up a care-giver center and would like to contribute substantial amounts of money, but because of the drain, this type of a drain on this disease, compared to heart—which is usually pretty quick and does not cost the family that much—cancer, in some cases, is quick; other cancer, of course, is long-term suffering and expensive to the families.

That might be one thing that I would hope that whoever is involved would look into maybe some different types of financing to further the various things that are in this report and that I am sure will come out of the report that our subcommittee will give.

I would like to ask you, Dr. Eisdorfer, one thing I never have understood, and everybody has talked about it for 2 days now, is the good days and the bad days. I am aware of the autopsy reports and things in several cases that I have seen, but why the good days and the bad days?

Dr. EISDORFER. If I consider myself good company, congressman, I think you are in good company. I do not know. Let me back off. We are learning an enormous amount about Alzheimer's disease, from the time 5 or 6 years ago when everybody though senility was a normal stage of aging, recognizing the disease. It was still a long way from knowing whether indeed we even have one disease. I alluded to this earlier.

One of my fellows is now very busy, going back and looking at the so-called microcirculation. Although we were in the original group that separated out vascular disease from Alzheimer's disease, we may now wind up putting microcirculation back into Alzheimer's disease. There may be a circulatory issue. I say that because one of the characteristics of the multi-infarct dementia, the other large group of patients with dementia—that is why ADRDA [Alzheimer's Disease and Related Disorders Association], a big single piece is the microstrokes, the ministrokes. As a matter of fact, one of the physicians here I think mentioned a patient who was thought to have Alzheimer's disease but who wound up with multiple strokes. So it is a misleading disagnosis.

It is conceivable that microcirculation may vary from day to day. We know that the catecholamine, the adrenaline noradrenaline that Dr. Davies talked about, which may play a role in certain kinds of Alzheimer's disease—that is where I did my initial research—that does vary from day to day, and it varies from time of day. We now think that it is highest, for example, in males at about 4 to 6 o'clock. Some people talk about the "sundowner's syndrome." Alzheimer's patients very often get agitated in the early evening, which we used to think was related to the changing light and the disorientation. It may be related to an internal chemical clock.

Alzheimer's patients are not devoid of memory. It is a terrible mistake. They may not respond verbally, but clearly they are, at some level or other, very much aware of what is going on. We kept Alzheimer's patients in our family group sessions—this goes back to 1974, 1975. It made a difference in the way they worked at home and so on. So they may be picking up extraneous material that we do not know about, and it is causing them to react in certain kinds of ways. Maybe Dr. Rabins can answer better than me.

Dr. RABINS. No, I do not have a better answer. I think these questions are very important to ask. Just as Dr. Marotta said, there are several different potential explanations for the fact that RNA levels seem to be different in Alzheimer's victims and in normal people. I think there are a lot of research studies that could be done to try to understand this sort of thing, looking at the levels of different chemicals in the blood, looking at how the person slept the night before, looking at what kind of stimulation they had that day, checking out their physical condition and their medication levels. There area whole variety of things.

The reason it is not just some kind of intellectual interest. The reason we would want to know that is because, maybe we can make a lot of days like the good days and diminish the number of bad days. I think we can answer those questions maybe sooner than we can find these ultimate answers.

Mr. WINN. You talked about the sleep problems in your testimony, and we heard a little bit of that yesterday. Of course, I think that is pretty well known a factor. At the same time—I think the panel before all of you—mentioned that as an Alzheimer's patient grows older, they grow away from drugs, which probably would include sleeping pills. How do you cope with a patient who maybe has taken sleeping pills from time to time to get sleep problems under control, but then develops an anti-sleeping-pill attitude?

Dr. RABINS. You are right. We first always try to avoid sleeping medicines. So if a person is not sleeping at night, the first things that we usually do are to make sure they are not napping in the daytime, to keep them active and busy. Again, this is one of the beauties of day care centers.

Mr. WINN. Yes, but to keep them from napping in the daytime when they are 80 years old or older, that is a little tough. It is tough enough trying to keep our committee awake.

Dr. RABINS. I have the same problem, especially after lunch. But this is the beauty, actually, of the day care centers. Here is a group of people who are experts at having lots of activities and changing things. It is extraordinary, the difference that these places sometimes can make, for example, in having somebody sleep. That is a nice inexpensive intervention, and you do not run the risk of the sleeping pill problems.

On the other hand, my experience is, on the majority of people you do have to try some kind of medicine. There are no good studies comparing all the different kinds of drugs. It is my experience that the usual sleeping pills that people take that are advertised are not good for the sleep problems in these patients. So this sort of thing needs to be looked at.

Dr. EISDORFER. I absolutely agree with Dr. Rabins on the hope to avoid drugs. What we do is get the patient up progressively early in the morning, make sure they get dressed and out of bed, where possible. We found that exercise—and a lot of older people just do not want to do exercise—but programming a long walk or even a short walk, and making exercise a routine, and again getting them up early is very important. The other thing we try to do, particularly in institutions where they get very rigid sometimes about bedtime, we try to convince the management and the nurses that maybe it is just as well if the patient stays up. Let's give them something to do so they won't wander around, night programs or evening programs, even if it is only watching television. And insisting that everybody lie in bed for long periods of time when they are not going to go to sleep, unless you medicate them, is wrong. If you medicate them in the evening, you pay a price in terms of the residual of the drug the next day. Patients are very sensitive to any kind of psychotropic medication.

Mr. WINN. Thank you very much. Thank you, Mr. Chairman.

Mr. GORE. Let me wrap it up with a couple of really short questions. If you could give me short answers, we would appreciate it. We are running out of time. We have a vote on the floor.

Dr. Rabins, is it safe to say that you highly recommend the idea of respite day care?

Dr. RABINS. Yes, sir.

Mr. GORE. When we look at the role of the country in trying to improve our response to this, we ought to give particular attention to respite day care?

Dr. RABINS. Yes, I would think so.

Mr. GORE. There is a proposal to establish nationwide uniform eligibility standards for Medicaid for Alzheimer's disease. Now they can be eligible in one State but not in another. Do you all think that is a sound idea, uniform Medicaid standards?

Dr. EISDORFER. I would think that would be reasonable. I think you need a preceding step. You need to define it better, and one of the problems is the diagnosis. We really do not have uniform standards for diagnosis; we just have a framework.

Mr. GORE. If you get an accurate diagnosis, then you think there should be——

Dr. EISDORFER. Sure. Then it would make sense.

Mr. GORE. And the absence of a brain biopsy at the present time means it is hard to get an accurate diagnosis.

Dr. RABINS. May I add one thing to that, if I may, really quickly? Perhaps we should consider, rather than focusing on making Alzheimer's disease specifically included or excluded, the functional level of the person. Wandering is a terrible problem to deal with, and that varies from patient to patient. I think I would prefer to see you focus on that.

Mr. GORE. We have a vote on the floor, and we would like to keep open the possibility of asking additional questions in writing to this panel. I want to thank you, and I want to thank the witnesses on all the panels today and yesterday and during the first day of the hearing in Johnson City, TN.

We will now do our best to absorb and digest and process all of the information, views, opinions, and recommendations given to us, and we will try to have a report which we hope will make a contribution. We have learned a lot and we have seen an awful lot of dedicated people, too. We appreciate all your work and your presence here.

With that, this hearing will stand adjourned.

[Whereupon, at 1 p.m., the committee recessed, to reconvene at the call of the Chair.]

APPENDIX

ADDITIONAL MATERIAL SUBMITTED FOR THE RECORD

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TESTIMONY BY

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for the

ALZHEIMER'S DISEASE HEARINGS
SUBCOMMITTEE ON INVESTIGATIONS AND OVERSIGHT
FOR THE
SCIENCE AND TECHNOLOGY COMMITTEE
UNITED STATES HOUSE OF REPRESENTATIVES

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October 4, 1984

Albert Gore, Jr., Chairman
Subcommittee on Investigations and Oversight
Committee on Science and Technology
United States House of Representatives
Washington, D.C. 20515

Dear Mr. Chairman:

I am a physician who has devoted his entire professional career to the care of the elderly and the education of health care professionals in geriatrics. From 1975 to 1979, I assisted in the development of one of the first geriatric medical residency and fellowship training programs in this country. Subsequently, I started the Division of Geriatric Medicine at the Mount Sinai School of Medicine and the Mount Sinai Medical Center in New York City. In 1983, I relocated my private practice of Geriatric Medicine to Huntington, New York.

A substantial portion of my practice is composed of patients who are suffering from Alzheimer's Disease and other memory loss problems. Because previous studies have shown that up to 40% of patients are misdiagnosed as having Alzheimer's Disease or other dementias, I believe it is important to understand the many causes of reversible or treatable memory loss that occur in older people. I have attached a discussion entitled "The Diagnosis and Therapy of Treatable Dementias in the Elderly" for inclusion in the record.

It is my pleasure to submit my testimony to the Committee.

Sincerely yours,

Fredrick T. Sherman, M.D.

FTS/jw
Encl.

The Diagnosis and Therapy of Treatable Dementias in the Elderly

by

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I. THE IMPACT OF DEMENTIA ON THE ELDERLY

Dementia is not a part of normal aging. While there are minor changes in recent memory during senescence, so called "benign senescent memory loss" of the elderly, there is no progressive global loss of intellectual function in the normal elderly person. About ten to fifteen per cent of those over the age of sixty-five have dementia. At age eighty, twenty percent of community residing elderly have some degree of dementia; conversely, eighty percent of eighty year olds have no dementia and have normal cognitive function.

Surveys of old people in hospitals and nursing homes reveal significant numbers with dementia. In a study of people over aged 70 admitted as emergencies to a general medical ward, twenty-five per cent had dementia and sixteen per cent had an acute confusional state on admission (1). A survey of in-patients over 70 years of age at a 400-bed medical-center affiliated community hospital found that nineteen per cent were mildly confused and another thirty-one per cent were moderately or severely confused. About one-third of all people over age 70 were admitted directly from a nursing or rest home (2). In nursing homes, it is generally accepted that fifty to seventy-five per cent of patients have impairment of mental function, the majority with some type of dementia.

II. POTENTIAL FOR REVERSIBILITY:

Dementia in the elderly should be considered to be "reversible," "arrestable," and/or "treatable" until proven otherwise. Numerous prospective and retrospective studies have shown that ten to thirty per cent of adult patients evaluated for dementia have a cause that is potentially arrestable or reversible (3,4,5). Since these studies were based on referrals to specialized neurology, psychiatry, neuropsychiatry or geriatric services, they reflect a highly selected group of patients, where many causes had been ruled out prior to referral. It is likely, therefore, that the primary care physician will see a greater prevalence of reversible dementias in his practice than indicated by these studies. The major cause for non-reversible, progressive dementia in late life is Senile Dementia of the Alzheimer's Type (SDAT). SDAT will not be discussed in detail. The reader is referred to recent references for specific details of etiology, diagnosis, and treatment strategies. (6,7,8)

III. DEFINITIONS

Impairment of intellectual function in the elderly can be either global, involving memory, judgment, abstract thought, language and a variety of other higher cortical functions, or be limited to any one of these. The syndromes of dementia and delirium are examples of global impairments, i.e. effecting multiple aspects of cortical function, while aphasia, apraxia, or amnesia are examples of disturbances of isolated functions.

Dementia is defined as a global impairment of intellectual function in a normally conscious, attentive, and responsive patient that is of sufficient severity to interfere with social or occupational functioning.(9) The terms chronic organic brain syndrome, OBS, chronic organic mental syndrome, OMS, chronic brain failure, chronic confusional syndrome, and senility are indiscriminately used in the literature. All these terms imply chronicity and irreversibility. As our knowledge and experience with dementia in the elderly has advanced, however, we have learned that dementia is not always irreversible nor is it necessarily chronic.

Impairment in at least three of the following five areas is needed to diagnose a dementia:

(1) memory, i.e., immediate, short term and long term;
(2) cognitive function, i.e., the ability to manipulate knowledge, including calculation, proverb interpretation, similarities and differences;
(3) visual spatial discriminiation e.g., impaired constructional ability such as difficulty drawing a clock and placing the dials at 3:30 or 11:10, or drawing a three dimensional figure such as a cube;
(4) language, e.g. fluent and nonfluent aphasias, impaired object naming; and
(5) personality, e.g. usually an alteration or exaggeration of premorbid traits with eventual deterioration.

IV. DIFFERENTIAL DIAGNOSIS OF ARRESTABLE OR REVERSIBLE DEMENTIAS:

The mneumonic, DEMENTIAS, can be utilized to remember the majority of the arrestable or reversible causes of dementia. (See Table I) Each etiology will be discussed separately, including diagnostic tests and specific therapies.

TABLE I

ARRESTABLE AND REVERSIBLE CAUSES OF DEMENTIA IN THE ELDERLY

D - DELIRIUM/ DRUGS

E - EARS/ EYES/ENVIRONMENT

M - METABOLIC/MULTI-INFARCT/MULTIPLE

E - EKG/ENDOCRINE/ETHANOL

N - NORMAL PRESSURE HYDROCEPHALUS / NUTRITIONAL

T - TRAUMA/ TUMOR

I - INFECTION

A - AFFECTIVE DISORDERS

S - SUBCORTICAL/SURGERY

DEMENTIAS

DELIRIUM:

Delirium is defined as a transient clouding of consciousness with an abrupt onset (hours to days) and of relatively brief duration (usually less than one month). It is characterized by disturbances in attention, memory, thinking, and perception (10). Fluctuation of consciousness i.e., lucid intervals when the patient thinks clearly, alternating with intervals of clouding of consciousness, is the hallmark of a delirium. Disturbances in the sleep-wake cycle as well as delusions and hallucinations may occur.

Older people, instead of presenting with the classic signs of delirium, may have a hypokinetic or quiet delirium characterized by withdrawal, decreased alertness and attentiveness, and daytime napping. These symptoms can exacerbate an existing dementia. The differential diagnosis of a delirium includes primary neurologic disease e.g., meningitis, cerebrovascular accident, and trauma, as well as many causes that lie outside the central nervous system, including acute infectious diseases, metabolic disorders, cardiopulmonary disorders, drug side effects and intoxications, fecal impaction, urinary retention, and accidental hypothermia or hyperthermia.

With a careful mental status evaluation, a new onset delirium can be differentiated from a dementia in the patient who is known to be demented. On the other hand, it may be impossible to determine how much of the clinical presentation is from dementia and how much from delirium when the two syndromes exist in the same patient seen for the first time. In general, one cannot diagnose a new dementia in the presence of a significant delirium. Screening tests: 1) vital signs including rectal temperature and respiratory rate. A respiratory rate greater than 25/minute may be the only sign of a pneumonia.; 2) physical examination including digital rectal examination and percussion of bladder; 3) metabolic-electrolyte screen including serum sodium, potassium, blood urea nitrogen, calcium, glucose, and liver function tests; 4) drug screen e.g. alcohol, digoxin, quinidine, theophylline, and/or dilantin blood levels; 5) complete blood count with differential. Note that the white blood cell count may be within normal limits but a left shift may be the only sign of an infectious process in the elderly.

DRUGS:

Many commonly used drugs in the elderly patient have the potential to effect mental status because of altered pharmacodynamics and/or pharmacokinetics. Altered pharmacodynamics lead to an increased central nervous system sensitivity to many drugs. Altered pharmacokinetics lead to a higher serum drug concentration, a prolonged drug half life, and/or a lowered metabolic clearance rate due to decreased hepatic biotransformation and/or renal excretion of many drugs. Specific categories of drugs that can cause an altered mental status that simulate or worsen an existing dementia are listed in Table II. These can easily be remembered because they are all members of the "anti-" family. See Table II below:

TABLE II

DRUGS THAT CAUSE OR EXACERBATE DEMENTIA IN THE ELDERLY

CATEGORY	EXAMPLES (brand names)
Anti-acid	Cimetidine (Tagamet)
Antianxiety	benzodiazepines (Valium, Librium, Serax, Xanax)
Anti-arrhythmia	Digoxin (Lanoxin) Quinidine (Quinaglute) Disopyramide (Norpace) Lidocaine
Antibiotics	Gentamicin, Tobramycin Nalidixic acid (Negram) Penicillin Rifampin (Rifadin)
Anticholinergics	benztropine (Cogentin) trihexyphenidyl (Artane)
Antidepressants	Polycylic (Elavil, Sinequan, Ludiomil) and other antidepressants (Desyrel)
Anti-diabetic	chlorpropamide (Diabinase)
Anti-glaucoma	Beta-adrenergic blockers (timolol malate, Timoptic)
Antihypertensives	Sympatholytics (Aldomet, Catapres) Beta-blockers (Inderal, Lopressor)
Anti-inflammatory	Nonsteroidal anti-inflammatory (Indocin, Motrin, Naprosyn and others) Steroids

TABLE II (cont)

Antiparkinsonian	levodopa (Laradopa, Sinemet) bromocriptine (Parlodel)
Antipsychotics	haloperidol (Haldol) thiothixene (Navane) thioridazine (Mellaril)
Antiviral	amantadine (Symmetel)
MISCELLANEOUS	Theophylline (Theodur)

Any of the "antis" implicated in causing a delirium or dementia can be given in a reduced dosage or can be discontinued altogether and the problem e.g., diabetes, hypertension, osteoarthritis or depression, monitored. Even digoxin, taken for years, can safely be withdrawn in many elderly patients who have had a history of congestive heart failure and are now compensated without cardiomegaly and in normal sinus rhythm .

Certain drug-drug interactions, in which each of the drugs has either altered pharmacokinetics and/or pharmacodynamics in the elderly, are particularly prone to lead to central nervous system side effects simulating or exacerbating a dementia, or causing a delirium. (See Table III).

TABLE III

DRUG-DRUG INTERACTIONS IN THE ELDERLY

THAT CAN EFFECT MENTAL FUNCTION*

DIGOXIN & QUININE, & QUINIDINE	increased DIG EFFECT leading to digoxin toxicity
CIMETIDINE & DIAZEPAM, & CHLORDIAZEPOXIDE	increased sedation
& PROPRANOLOL	increased BETA BLOCKING effect
& THEOPHYLLINE	increased THEOPHYLLINE TOXICITY
ETOH & BENZODIAZEPINES	increased SEDATION
PROPRANOLOL & THEOPHYLLINE	decreased THEOPHYLLINE CLEARANCE & TOXICITY
and TIMOLOL MALATE (eye drops)	worsening of congestive heart failure or onset of bronchospasm
METHYLDOPA & HALDOL & LITHIUM	HALDOL TOXICITY LITHIUM TOXICITY

*either or both of the drugs has been shown to have altered pharmacodynamics or pharmocokinetics in the elderly.

If a drug or a drug-drug interaction can be implicated as a cause of the dementia or delirium, then reduction, withdrawal, or a change in dosage regimen should be performed to see if the mental changes resolve.

In addition, medication errors due to poor comprehension and/or compliance with a medication regimen can lead to worsening of a dementia or a delirium by exacerbating an underlying medical problem, such as congestive heart failure or chronic obstructive lung disease. Screening tests: 1) serum drug levels e.g., quinidine, digoxin, or theophylline; 2) drug reduction or discontinuation, and monitoring.

DEMENTIAS

EYES AND EARS:

Sensory deprivation from decreased vision and/or decreased hearing may make an elderly person appear to be demented. The removal of a cataract or the use of low vision aids as well as the removal of cerumen, or the use of a hearing aid, for example, may improve mental status. Screening tests:

a) visual acuity by Snellen eyechart and visual fields by confrontation;
b) fundoscopic examination;
c) otoscopic examination;
d) Weber and Rinne tests;
e) audiometric testing to determine the type(s) (sensorineural and/or conductive) and the extent of hearing loss, and the potential benefit from a hearing aid;
f) examination of the hearing aid to make sure that the battery is functioning and the tubing is not occluded by cerumen.

ENVIRONMENT: Elderly people transferred from their own home to a nursing home or hospital, or from a hospital to a nursing home may have a worsening of their dementia because of unfamiliarity with their new environment and loss of valuable cues, such as familiar faces, pictures and furniture, as well as inadequate lighting. Seemingly small gestures, such as bringing familiar pictures, furniture or a bedspread, to a well lit room when the older patient moves to a nursing home may prevent or decrease confusion. "Sundowning", the increased confusion that occurs in demented patients during the evening when visual and auditory stimuli decrease, can be dealt with by many of these same approaches.

DEMENTIAS

METABOLIC:

Because older people have a tendency to secrete increased amounts of anti-diuretic hormone and because they cannot excrete water and salt as effectively as younger adults, they are prone to dehydration and hyponatremic and hypernatremic states. Azotemia, uremia and acid-base disturbance can also effect mental status. Screening tests: serum sodium, chloride, bicarbonate, potassium, creatinine, and blood urea nitrogen.

MULTI-INFARCT:

Multi-infarct dementia (MID) is the main cause of 10-20% of all dementias and a contributory factor in approximately 10-15%. MID results from multiple small cortical strokes occurring at different times. MID is characterized by an abrupt onset followed by a step wise deterioration in intellectual functioning, as contrasted to the slow, often insidious, progressive deterioration seen with Alzheimer's disease, or other dementias. MID is usually associated with focal neurological symptoms and signs and occurs more commonly in men with hypertension. Hemiparesis, a small stepped gait, asymmetric and extensor plantor reflexes and pseudobulbar palsy, characterized by emotional lability, dysarthria, and dysphagia, are commonly seen. The intellectual deficits are "patchy" depending on which areas of the brain have been affected. MID may be arrestable if associated hypertension, diabetes, and/or cardiac dysrhythmias, especially new onset atrial fibrillation, are treated. Electrical or chemical cardioversion, however, is unlikely to be successful in elderly people with chronic atrial fibrillation with an enlarged left atrium. Oral anticoagulation, while probably decreasing cerebral emboli, carries an increased risk of bleeding in the elderly and requires close monitoring with strict compliance by the patient, often with the help of a caregiver or family member. Screening tests: Neurological examination, computerized axial tomographic (CAT) scan of the head.

MULTIPLE: Multiple causes of dementia must be considered when a patient presents for the first time or when an existing dementia worsens. For example, the 85 year-old man with senile dementia of the Alzheimer's type (SDAT), may develop hypothyroidism, acute urinary obstruction secondary to prostatic hypertrophy, or a medication side effect, which worsens his dementia. Treatment of the hypothyroidism, urinary drainage, or alteration of the medication may return the patient's mental status to its previous state.

DEMENTIAS

ENDOCRINE:

Hypercalcemia secondary to primary hyperparathyroidism, multiple myeloma, thiazide diuretics, vitamin D intoxication, or metastatic cancer can cause a dementia. Hypocalcemia, recurrent hypoglycemia, and hyperglycemia, with and without acidosis, can cause or exacerbate a dementia. Hypothyroidism and hyperthyroidism, either classic or apathetic, are also causes of an arrestable or reversible dementia. Screening tests: a) serum Ca, phosphorus and alkaline phosphatase; b) serum T_4 and if low or high, thyroid binding globulin, TSH, or T_3 resin uptake; c) plasma glucose; d) chest x-ray.

EKG:

Cardiac causes for dementia include dysrhymias, multiple cerebral emboli and/or heart failure, all of which can lead to decreased cerebral profusion and cerebral anoxia. "Cardiogenic dementia" resulting from episodes of anoxia due to cardiac dysrhymias should be suspected in the elderly patient with lightheadedness, syncope, bradyarrhythmias, tachyarrhythmias, or sick sinus syndrome.(11,12)

Since the standard 12-lead electrocardiogram may not show any abnormalities in the elderly patient with abrupt onset of dementia, with or without focal neurological symptoms or signs, it may be necessary to perform 24-hour cardiac monitoring to determine whether any dysrhymias may be causative. Screening test: 24-hour cardiac monitoring.

ETHANOL:

Chronic alcohol ingestion can cause a dementia that may be exacerbated by portosystemic encephalopathy or Wernicke-Korsakoff syndrome. Alcoholic dementia is usually associated with other evidence of chronic alcohol abuse including peripheral neuropathy or cerebellar degeneration. Discontinuing alcohol ingestion over a prolonged period of up to five years improves mental function.(13) Screening tests: alcohol intake history; liver function tests, including SGPT, SGOT, and bilirubin.

DEMENTIAS
NUTRITIONAL:

B_{12} deficiency and folic acid deficiency can present either as an isolated dementia or in association with other neurological and psychological findings, including subacute combined degeneration. (14,15) Both deficiency states can occur with a normal hemoglobin and normal red blood cell indices. Parenteral treatment for the neurologic complications of B_{12} deficiency, including dementia, is more intensive than the treatment for the hematologic complications. B_{12} therapy may halt the progression of the dementia or, less likely, reverse it. The recommended treatment for patients with neurologic complications varies. I prescribe 1000 micrograms of Hydroxocobalamin given intramuscularly every other day for one month and then every two weeks for six months. Usually a family member can be taught to give intramuscular injections. Maximal neurological improvement usually occurs during the first six months, but there may be improvement up until one year of therapy. After maximum recovery or one year has elapsed, 1000 micrograms can be given monthly for life. Screening tests: Serum B_{12} and red blood cell folate levels.

NORMAL PRESSURE HYDROCEPHALUS (NPH):

NPH is a syndrome characterized initially by an unsteady gait, often with falling, followed by a mild to moderate dementia and finally urinary incontinence, probably secondary to an uninhibited neurogenic bladder. There is enlargement of the ventricles, a normal cerebrospinal fluid pressure and little or no atrophy of the cortex. Pathophysiologically, NPH is caused by the impaired absorption of the cerebral spinal fluid by the arachnoid villi. (16) NPH can be primary (idiopathic), or be secondary to meningitis, head injury or subarchnoid hemorrhage. Ventriculoperitoneal shunting of the cerebrospinal fluid can lead to partial or total improvement in idiopathic NPH in about fifty per cent of cases. (17) Screening tests: a) computerized axial tomographic (CAT) scan of the head, if the history suggests NPH; b) ^{111}Indium cisternography if the CAT scan and history are suggestive; c) test removal of 20-30 ml of cerebrospinal fluid by lumbar puncture may induce clinical improvement.

DEMENTIA
TRAUMA:

An obvious head injury can cause a subdural hematoma or a post-concussion dementia. On the other hand, the fall or head injury may not be reported or obvious from physical examination.

Neurological evaluation may not reveal any focal deficit with a subdural hematoma.(18) Elderly patients with head injury only may recover cognitive function over months to years after the incident. (19) Screening test: Neurological examination; CAT scan of head.

TUMOR:

Gliomas, primarily glioblastomas, account for about fifty per cent of all CNS tumors in the elderly. Metastatic tumors from lung, breast, colon, kidney, and prostate are responsible for about twenty-five to thirty-five per cent.(20) Meningiomas account for about twenty per cent. In one series, approximately fifty per cent of selected elderly people with brain tumors improved after craniotomy for total or partial tumor resection. (21) Screening tests: chest x-ray, CAT scan of head, EEG, cerebrospinal fluid analysis.

DEMENTIA

INFECTION:

While tuberculosis and endocarditis can both cause a chronic dementia, these are rare. Symptomatic neurosyphilis, specifically general paresis, can present as a dementia. A history of syphilis may be forgotten, denied, or never known, and past treatment records are generally unavailable. The serologic antibody tests for syphilis e.g., VDRL or RPR, are inadequate as screening tests for neurosyphilis. (22,23) False-negatives are common in older patients with neurosyphilis. In addition, a positive VDRL in low titer may be a result of increased non-specific antibody formation seen in the elderly. Consequently, a serum FTA-ABS should be ordered on all patients with a newly diagnosed dementia. If the serum FTA-ABS is positive on two occasions, then an analysis of the cerebrospinal fluid should be made for pleocytosis, protein and VDRL. If these are consistent with neurosyphilis, the demented patient should be treated. If the patient and/or family refuses a lumbar puncture for cerebrospinal analysis, the demented patient with a positive serum FTA-ABS on two occasions should be assumed to have neurosyphilis and treated. Screening tests: serum FTA-ABS, serum VDRL or RPR.

DEMENTIA

AFFECTIVE:

In older people, depression can mimic dementia and vice versa.(24)In addition, the demented elderly patient may become depressed, and the depressed patient may develop a superimposed dementia. The elderly patient with a stroke or multi-infarct dementia may develop a post-stroke depression.(25,26) In either case the depressive component may be treatable with antidepressant medication and/or electroconvulsive therapy. Generally, the depressed elderly patient complains of memory loss of recent onset, rather than the gradual, poorly dated onset of memory loss that occurs with senile dementia of the Alzheimer's type. The elderly patient with depression feels and acts his best in the afternoon and evening as opposed to the patient with Alzheimer's disease who may become disoriented and confused (i.e., "Sundowning") in the evening and afternoon. The patient with depression may have vegetative signs, such as sleep disturbances, weight loss and constipation. It should be noted, however, that these three signs are common amongst elderly people, in general, as well as elderly demented people. At present, neither the overnight dexamethasone suppression test nor the response of thyroid stimulating hormone (TSH) to thyrotrophin-releasing hormone (TRH) has been well studied in elderly patients with cognitive impairment associated with dementia and/or depression. Consequently, they cannot currently be recommended as clinical tools to differentiate depression from dementia in the elderly, or to monitor or predict response to treatment. (27-32) If a depression is suspected as the sole cause or a contributing cause to cognitive impairment, then the patient should be given a therapeutic trial of treatment e.g., antidepressants or electroconvulsive therapy, with psychotherapy.

DEMENTIAS
SUBCORTICAL:

Subcortical dementias include those caused by Huntington's disease, Wilson's disease, progressive supranuclear palsy, and Parkinson's disease. (33) While both of the latter two occur in the elderly, it is only the dementias associated with Parkinson's disease that should be addressed when speaking of arrestable and treatable causes of dementia in the elderly. Subcortical dementias differ from cortical dementias (e.g., Alzheimer's and Pick's disease) in many ways. In subcortical dementias, the predominant feature is extreme slowness of movement characterized by a slow gait, stooped posture, disheveled appearance, forgetfulness, and apathy. While the patient with a subcortical dementia may have a dysarthria or hypophonic speech, their language function is generally intact. Elderly patients with Parkinson's disease may develop evidence of dementia and delirium. (34-39) This may be secondary to: 1) medication side effects from anticholinergics or levodopa preparations; 2) onset of depression causing cognitive impairment; 3) the development of a dementia from any cause, including Alzheimer's disease or multi-infarct disease; and/or 4) the natural history of Parkinson's disease. Clearly, diagnosis and treatment of the first three may result in clinical improvement and reversibility. Screening tests: lowering dosage of anticholinergic and/or levodopa preparations.

SURGERY:

It is well appreciated that elderly patients may develop a worsening of a dementia or a new onset of delirium post-operatively. An agitated delirium usually presents by the third post-operative day and lasts one to two days. Cataract extraction, with and without a lens implant, may lead to postoperative confusion. While it is commonly believed that sensory deprivation after cataract extraction leads to confusion, other factors such as infection, metabolic causes, drugs, cerebral hypoxia, new onset stroke, myocardial infarction, pulmonary embolism and heart failure must also be considered as they are all treatable. If no cause is found, judicious use of analgesics, major tranquilizers in low dose, and environmental stimulation by staff will usually calm the patient.

With increasing numbers of older people undergoing cardiac surgery for valve replacement and coronary artery bypass, careful pre and post operative assessments of neurological and mental status must be performed because of the high prevalence of neurological abnormalities in the immediate post operative period, particularly after the use of extracorporeal circulation. Motor and sensomotor hemisyndromes are common at ten days post operative. One year after cardiac surgery involving extracorporeal circulation, cognitive disorders accounted for sixty per cent of all neurological sequelae. (40) While the etiology of these sequelae are uncertain, it is important for the clinician to be aware of impaired cognitive function that may occur after extracorporeal circulation in the elderly. (41, 42)

CONCLUSION

The evaluation of the confused elderly patient, particularly in the ambulatory setting, will yield a potentially arrested or reversible cause in up to a third of patients, prevent unnecessary and prolonged hospitalization, and decrease the need for nursing home placement and costly homecare. The mneumonic, DEMENTIAS, will assist the clinician in performing a thorough evaluation on every elderly patient who is suspected of having dementia.

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STATEMENT FOR THE RECORD

SUBCOMMITTEE ON INVESTIGATIONS AND OVERSIGHT OF THE HOUSE COMMITTEE ON SCIENCE AND TECHNOLOGY

HEARINGS SEPTEMBER 19 AND 20, 1984

STATEMENT SUBMITTED BY
DISTRICT OF COLUMBIA SPEECH-LANGUAGE-HEARING ASSOCIATION
PRESIDENT
SCOTT BALLY, M.S.
GOVERNMENTAL AFFAIRS COMMITTEE
WENDY FOX, M.S.
VIRGINIA GEOFFREY, PH.D.
GLORIA NELSON, M.S.
LIZ RICHARDSON, M.S.

We are pleased that the Subcommittee on Investigations and Oversight has recognized the concerns of the community about the illness of Alzheimer's disease. From our perspective we would like to emphasize the communication needs of a person with Alzheimer's disease:

* The importance of timely evaluations of communication skills to meet the changing needs of the person with Alzheimer's disease

* The need for therapy to teach compensatory communication strategies to help the person with Alzheimer's disease function more independently

There are one and a half million Americans with Alzheimer's disease. Essentially all of these people have or can expect to have communication problems during their lifetime.

With proper care most persons with Alzheimer's disease continue to live and function with their families in a communicative environment for many years. The quality of their care and well-being is directly influenced by (1) their families' understanding of the communication disorder and its progressive change, and (2) the person's ability to compensate for his or her communication disorder.

Under the current laws, people diagnosed as having Alzheimer's disease cannot receive any reimbursement from Medicare for any type of speech, language, or hearing therapy, while people with similar degenerative diseases, such as Parkinson's disease, multiple sclerosis (MS), and amyotrophic lateral sclerosis (ALS) are eligible for reimbursement of these services.

We are writing as concerned individuals and service providers who know of the serivces that could be available for the person with Alzheimer's disease. We believe that a higher quality of life could be maintained by maximizing communication skills at all levels of disability. The longer communication skills remain useful, the longer the patient can live at home or in a nursing home with some independence and dignity.

By not being covered by Medicare, persons with Alzheimer's disease are effectively denied speech, language and audiological services that they need. We feel that these persons have the same rights to service as those in hospice care, or those with other similar neurological diagnoses and urge reexamination of the rationale for excluding Alzheimer's disease. Thank you.

PLAN FOR THE GOVERNOR'S TASK FORCE
ON ALZHEIMER'S DISEASE AND RELATED DISORDERS
STATE OF MARYLAND

According to the U.S. Bureau of the Census, there are for the first time more Americans over sixty-five than teenagers. Presently, Alzheimer's disease and related disorders are affecting between 5 percent and 10 percent of all people over the age of sixty-five. There is no identified cause or known cure for this devastating disease.

The General Assembly of Maryland and the Governor of Maryland recognized that this problem will become more common and take even greater toll as Maryland's aging population continues to grow. Recognizing that fact, Governor Harry Hughes established a Task Force on Alzheimer's Disease and Related Disorders.

The Task Force was asked to collect data on incidence, prevalence, and geographic distribution and to make recommendations to improve the availability of such data; to identify available resources and gaps in needed services for persons suffering from this disease and related disorders, and their families, and to make recommendations regarding services, support systems, and funding mechanisms which may alleviate these needs; to promote and provide educational programs for health professionals concerning diagnosis, resources for management, and documentation of Alzheimer's disease and related disorders; to develop a network of health professionals throughout the State with special interest and expertise in the diagnosis and management of persons with Alzheimer's disease and related disorders to be a resource for counseling caregivers and consultation to other service providers; to provide public education in order to increase understanding and awareness of the disease and related disorders, and of resources available to assist the affected persons and their families; and finally, to encourage research into the causes and effective treatment of Alzheimer's disease and related disorders.

To carry out the mission of the Task Force on Alzheimer's Disease and Related Disorders, five committees were established. These included:

1) Demography and Data;
2) Services and Resources;
3) Funding and Finances;
4) Training and Education;
5) Research.

For the sake of practicality, the Committees on Services and Resources and Finances and Funding chose to work together because of overlap of Committee members and also because of the commonality of purpose.

GOVERNOR'S TASK FORCE ON ALZHEIMER'S DISEASE AND RELATED DISORDERS

Demographics and Data Committee

Chairperson: Patricia M. Smith, M.D., M.P.H.
Edmund Beacham, M.D.
Patricia Lusk, R.N.
Thomas R. Price, M.D.

Finance and Funding Committee

Chairperson: Fred M. Gloth, Jr.
Rosalie S. Abrams (Frank Furstenburg)
Honorable Clarence W. Blount
David F. Chavkin
Jacqueline D.F. Fassett
Lloyd F. Hoover

Research Committee

Chairperson: Thomas R. Price, M.D.
Glenn Kirkland
William Reichel, M.D.

Resources and Services Committee

Chairperson: Honorable Peter G. Callas
David F. Chavkin
Jacqueline D.F. Fassett
Glenn Kirkland
Kay Mehlferber, R.N.

Training and Education Committee

Chairperson: Nancy Mace
Rosalie S. Abrams (Shirley Whitfield)
Edmund Beacham, M.D.
Honorable Peter G. Callas
Patricia Newton, M.D.
William Reichel, M.D.

The Committee on Demography and Data will determine the prevalence of Alzheimer's disease and related disorders as a basis for planning services and their reimbursement. Information will be collected by diagnosis; by geographic region; by socioeconomic status and race; by living arrangements; and by other parameters. There will be an attempt to identify gaps in data and the effect of data gaps on planning.

Our knowledge concerning the demography of Alzheimer's disease and related disorders is greatly influenced by physician understanding which affects clinical diagnoses and diagnoses on death certificates. We can turn to the original Newcastle study in Great Britain, the New Haven study, data from nursing homes, death certificates, census data, state hospital data, ADRDA membership, ECA data and other sources. One possibility is to establish new death certificate guidelines and to educate physicians regarding this system.

ACTIONS:

The Committee will:

1) Determine which studies exist currently.

2) Ally the Committee with the national study being conducted by Secretary Margaret Heckler.

3) Design and request studies appropriate to facilitate the work of other committees, and coordinate data requests between committees.

4) Request data from Blue Cross and Blue Shield of Maryland and Medicare and Medicaid.

5) Request data from the Veterans Administration.

6) Identify federal initiatives in demography and collection of data.

7) Identify the diagnostic criteria used by physicians in Maryland regarding Alzheimer's disease and related disorders.

8) Identify information pertaining to DRG's and Alzheimer's Disease and related disorders.

9) Obtain ECA data.

(Contact: Dr. Morton Kramer at Hopkins; Dr. Jacob Brody at National Institute on Aging).

10) Obtain current state hospital data.

(Contact: Dr. Tom Krajewski and Dr. Patricia Smith of State Health Department).

11) Obtain nursing home data.

(Contact: Nancy Mace).

12) Obtain data pertaining to acute hospital admissions from the Health Services Cost Review Commission; obtain data from Maryland Health Resources Planning Commission.

13) Obtain study dealing with frequency of diagnosis in the Emergency Room.

(Contact: Gene Floyd).

14) Obtain information regarding geriatric evaluation services and day care. Data has not been collected in the past, but it is possible to begin collection of such data.

15) Obtain census data.

SPECIAL INITIATIVES OF THE COMMITTEE ON DEMOGRAPHY AND DATA

1) Improve death certificate reporting of Alzheimer's disease

a) by professional education to physicians; and

b) exploring possibility of change in death certificate guidelines to ensure identification of Alzheimer's disease among causes of death.

2) Improve the accuracy and frequency with which Alzheimer's disease is reported as the leading diagnosis in the acute hospital setting in order to make data of the Health Services Cost Review Commission more reliable as to the prevalence of Alzheimer's disease and incidence of acute hospital admissions caused by this disease.

a) Professional education to medical record librarians as far as coding of Alzheimer's disease, related disorders, and other organic brain diagnoses.

3) Consider, plan and implement a state autopsy network.

(Contact: Dr. Peter Whitehouse at Hopkins; June White in Minnesota)

4) Promote the development of an educational package related to the diagnosis of Alzheimer's disease and related disorders to be distributed to Maryland physicians in Family Practice, Internal Medicine, Psychiatry and Neurology.

The Committees on Services and Resources and Funding and Finances will identify available services and resources and gaps in these services and resources, and make recommendations regarding services, support systems, and funding mechanisms which may alleviate the gaps which currently exist.

CURRENT STATUS OF THIS GOAL

Activities and information relevant to the stated goals do exist partially. However, a focused, coordinated, systematic and coherent approach is needed.

A. Development of a comprehensive inventory of the needs of patients and families, taking into account social, psychological, environmental and other needs as well as geographic differences within the state.

1) Needs of patients:
   - a) Diagnostic
   - b) On-going physician support
   - c) Supportive environment
   - d) Social environment
   - e) Psychological support

2) Needs of families:
   - a) Information
   - b) Respite
   - c) Peer support
   - d) Financial assistance

(Contact: Dr. Reichel -- Report of the AMA/ANA Joint Task Force on the Improvement of the Health Care of the Elderly).

B. Identification of currently available resources at the institutional and noninstitutional levels.

1) Services and resources now available (parameters):

a) Provider: government, private sector, voluntary.

b) Kinds of, e.g., medical respite, information and referral, residential.

c) Characteristics of, e.g., geographic location, accessibility, responsiveness, level of sophistication, utilization of, coordination with others, training of staff.

2) Services and resources needed but not available or where availability is limited:

a) Provider.

b) Kinds of.

c) Characteristics of. Will include identification of those parameters which Task Force believes are the most needed.

(Contact: Dr. Reichel -- Report of the 1981 White House Conference on Aging).

C. Identification of currently available public and private sector funds and sources of financial support such as third-party payment and tax incentives.

1) Cost (parameters):

a) Current costs, cost projections.

b) Costs of providing care, service; disability payments; costs of lost wages.

c) Who incurs costs, e.g., the government, the caregiver, etc.

2) Funding sources now utilized (parameters):

a) Provider, e.g., government, private sector, volunteer.

b) By geographic region.

c) By type of care, e.g., medical, information and referral, respite.

d) Gaps in funding.

e) Equity of funding.

3) Funding needed or recommended.

D. Identification of gaps in resources, services, funding and financial support.

E. Recommendations.

1) Public and agency.

2) Legislative.

ACTION OR ACTIVITIES NEEDED TO CARRY OUT GOALS:

A. Utilizing the five HSA areas of the state, the Committees propose the development of a series of regional hearings in order to affirm and add to data collected regarding needs, resources, gaps in services and funding.

B. Data relevant to the stated goals will be listed from the following groups and/or organizations:

1) Offices on Aging
2) Maryland Nursing Home Association
3) Maryland Adult Day Care Association
4) American Hospital Association
5) United Way Agencies
6) Insurance industry; Medicare; Medicaid
7) State insurance commissioner
8) Secretary of Maryland Department of Human Resources

9) VA Hospital and other facilities

10) Maryland chapter of the American Nurses Association

11) Medical and Chirurgical Faculty of the State of Maryland

12) Proprietary home care agencies, e.g., Voluntary Nurses Association, Upjohn, etc.

13) Self-help organizations

14) Council of Churches

15) Associated Catholic Charities

16) Lutheran Social Services

17) Associated Jewish Charities

18) Maryland Chapter of the National Association of Social Workers

19) Maryland Division of Labor and Industry

20) Secretary of Maryland Department of Health and Mental Hygiene

21) National Institutes of Health

22) National Institute of Mental Health

23) Alzheimer's Disease and Related Disorders Association, Inc., both the national office and state chapter

24) Department of Health and Human Services, Social Security Administration, Health Care Financing Administration

C. Analysis and synthesis of data from survey and hearings.

D. Preparation of recommendations based on data analysis.

SPECIAL INITIATIVES OF THE COMMITTEES ON SERVICES AND RESOURCES AND FUNDING AND FINANCES:

1. Build a network of physicians and other care providers surrounding each population center in the state to act as consultants to other health care providers in the care of patients throughout the state. These

designated regional consultants would receive appropriate training from the academic specialists from the University of Maryland, Johns Hopkins Hospital, and selected community hospitals.

2. Explore reimbursement by third-party payors for "custodial care" in mental hospitals or other appropriate sites when those sites are the sole source of care adequate to the patient's need.

   a) Develop a fiscal note based on various per diem costs and probable numbers of persons requiring this care in relation to each of the major third parties who would be responsible for this coverage.

3. Promote the establishment of special care units which may be day care or inpatient care at various levels of intensity, such as chronic hospital and nursing home. These care settings would provide both the psychogeriatric and hands-on nursing care which are needed by many elderly dementia patients.

4. Create a directory of services and resources in Maryland which are available to victims of Alzheimer's disease and their families and caregivers.

5. Promote the establishment of a new interagency coordinating council to coordinate the state government's many activities relating to Alzheimer's disease and related disorders, and report annually to the governor and the legislature on these activities and priorities for the future.

The Committee on Research will:

1. Identify the magnitude and nature of the research being done in the state of Maryland. This includes research of the following types: basic or fundamental; demographic; epidemiological; psychiatric; psychological; social; family; and therapeutic.

2. Identify primary areas for promising research particularly needed in the state of Maryland.

3. Identify funding sources, e.g., federal, state and private.

4. Identify what is currently being done nationally and how it affects Maryland.

5. Survey both the current status of research in Maryland and the projected status. What has been successfully funded in Maryland? What proposals have not been funded in Maryland?

6. Identify potential research that should be funded by the state of Maryland.

7. Identify research that is unique to this state, such as population groups.

8. Identify areas that the state of Maryland can urge greater federal funding.

9. Identify uncovered areas of research in the state of Maryland, both basic and applied.

ACTIONS:

Develop survey instrument and send to the following resources:

A. Johns Hopkins University School of Medicine and Johns Hopkins School of Hygiene and Public Health

B. National Institutes of Health

C. University of Maryland School of Medicine

D. University of Maryland College Park

E. Armed Forces Medical School

F. Veterans Administration

G. Gerontology Research Center, particularly the Longitudinal Study at Baltimore City Hospitals

H. Fort Detrick and Edgewood Arsenal

Analyze surveys.

Write report and develop recommendations.

SPECIAL INITIATIVES FOR THE COMMITTEE ON RESEARCH:

1) The State of Maryland might want to set up a model training curriculum on Alzheimer's disease and related disorders for nurses and then develop a test to determine the effectiveness of the training.

2) Ask ADRDA to provide a succinct summary of the present status of research nationally to be included as an appendix to the Task Force report.

The Committee on Training and Education:

There is a fundamental need for education and training at all levels. Currently existing training and education programs will be identified.

The Committee will:

1) Identify what training and educational programs exist;

2) Identify the future plans of organizations and educational institutions for training and education in this area;

3) Identify what is believed to be needed by a variety of organizations, agencies and educational institutions.

The enclosed educational questionnaire (see ADDENDUM) will be sent out to selected agencies, organizations and institutions. It will also be sent out with all other surveys which are utilized by the other committees.

SPECIAL INITIATIVES OF THE COMMITTEE FOR TRAINING AND EDUCATION:

1. Build a network of physicians and other care providers surrounding each population center in the state to act as consultants to other health care providers in the care of patients throughout the state. These designated regional consultants would receive appropriate training from the academic specialists from the University of Maryland, Johns Hopkins Hospital, and selected community hospitals.

2. Promote November to be designated in Maryland as National Alzheimer's Disease Awareness Month.

ADDENDUM:

Educational Questionnaire for four-year colleges; nursing schools; medical schools including the University of Maryland School of Medicine, Johns Hopkins School of Medicine, and the Armed Forces Medical School; social work schools; graduate schools; law schools; community colleges; state examination boards; and state associations for physicians, nurses, social workers, rehabilitative therapists, aides, lawyers, and judges.

1. What education/training do you offer regarding Alzheimer's disease?
    - Lecture
    - Full course
    - Required for whom
    - Elective for whom
    - Academic level:
        - Undergraduate
        - Graduate
        - Medical Student
        - Resident
        - Post-doctoral
2. Does this material include:
    - (a) Information about normal aging
    - (b) General information about abnormal aging
    - (c) Information relevant to the caregiver (impact, support for, family therapy)

(d) Information relevant to the community (social costs, public policy)

(e) Field placement/patient care

(f) Information about diagnosis

(g) Information about patient care/intervention/treatment

3. Do you have a plan for future courses/lectures on Alzheimer's disease? Please explain.

4. Do you think such course material is needed?

Lecture

Full course

Required for whom

Elective for whom

Academic level:

Undergraduate

Graduate

Medical Student

Resident

Post-doctoral

Does this material include:

(a) Information about normal aging

(b) General information about abnormal aging

(c) Information relevant to the caregiver (impact, support for, family therapy)

(d) Information relevant to the community (social costs, public policy)

(e) Field placement/patient care

(f) Information about diagnosis

(g) Information about patient care/intervention/treatment

If no, please explain.

5. Do you hold grants/have special funds for training in dementia?

Questions for public school system:

1. What education material on old age is included in the curriculum? At what level?

2. Information about Alzheimer's disease included in the curriculum? At what level?

3. What information do you think should be included and at what level?

Questions for service agencies and policy agencies:

1. What training/education has your staff received in Alzheimer's disease?
   - (a) Lecture (how many)
   - (b) Full course
   - (c) Required
   - (d) Elective
   - (e) CEU's
   - (f) In-service by staff
   - (g) In-service by outside professional
   - (h) On-the-job
   - (i) Reading material/literature
   - (j) Videotape
2. Does this material include:
   - (a) Information about normal aging
   - (b) General information about abnormal aging
   - (c) Information relevant to the caregiver (impact, support for, family therapy)
   - (d) Information relevant to the community (social cost, public policy)
   - (e) Field placement/patient care
   - (f) Information about diagnosis
   - (g) Information about patient care/intervention/treatment

3. Who has received education/training?

   (a) Administrative personnel

   (b) Professional direct service providers

   (c) Nonprofessional direct service providers

4. What training in dementia is required? Do you hold grants/funding for training in dementia?

5. Do you feel that training should be changed/upgraded? If so, please elaborate.

Questions for agencies offering education: (State Office on Aging, Maryland Consortia, Association for Higher Education in Gerontology (AGHE), Secretary Heckler's task force:

1. Enclose questionnaire and explain goals.
2. What training are you offering, planning, what do you feel is needed?

Questions for public or ADRDA:

1. In your contacts with policymakers/service providers/paraprofessionals, in what areas do you feel they need more training?
2. In what areas do you feel you need more education/training?